THE AMERICAN SURGEON

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Vol. 24, No. 10 October, 1958

Presidential Address*

ETERNAL VIGILANCE

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This being our tenth anniversary, it is appropriate that we review what has happened in the Southwest Surgical Congress, in organized medicine, and in the social structure of our country during these past 10 years. I also wish to alert you to what may happen to all strata of society and to our organization as well as to many similar organizations during the next decade.

The first thought of organization of the Southwestern Surgical Congress had its origin in a discussion between Dr. B. T. Beasley of Atlanta, Georgia, Dr. R. L. Sanders of Memphis, Tennessee, and Dr. Walter Stuck of San Antonio, Texas, during the annual meeting of the Southeastern Surgical Congress in Hollywood, Florida, in the spring of 1948. This discussion emphasized the fact that the great Southwest was the only region of the United States without a surgical organization of its own. Drs. Beasley and Sanders encouraged Dr. Stuck to explore the feasibility and desirability of developing such an organization. Dr. Stuck followed this conference by contacting well known surgeons in the then nine Southwestern Surgical states. Later, Wyoming was included in the area. Response from surgeons in all of the states was enthusiastic.

Inquiries as to the needs and desirability of the formation of such a society brought in another group of men who were making plans to develop a Southwestern Surgical Society of a more limited scope. This group was spearheaded by Dr. Louis Good of Texarkana, Texas, and Dr. H. G. Cogswell of Tucson, Arizona. These men and their friends contributed much to the organization of the Southwestern Surgical Congress.

Dr. Stuck, in his quiet but persistent manner, was responsible for the birth of the Congress. This required considerable work, including much correspondence during the period of development. Drs. Sanders, Beasley, and other members of the Southeastern Surgical Congress gave most freely of their time and were Dr. Stuck's guiding lights and inspiring helpers.

During the months that followed the initial inquiries in April, 1948, Dr. Charles Roundtree of Oklahoma City worked closely with Dr. Stuck and contributed much to the organization. Planning together, these men organized and crystallized the plans for the organizational meeting which was held in Oklahoma City on October 3, 1948. Then, with Dr. Stuck as chairman and Dr. Roundtree as secretary, the task of forming a definite surgical organization was undertaken. These two men, with the assistance of outstanding surgeons from the Southeastern Surgical Congress and men in the area of the Southwestern Surgical Congress worked diligently through the winter months, and by early spring of 1949 basic organizational meetings of the 10 states had been established and final plans had been completed.

On April 10, 1949, the society was organized and named the Southwestern Surgical Congress. The minutes of this meeting have been made a part of the historical record. Dr. Walter Stuck was elected the first president and Dr. Charles Roundtree the first secretary-treasurer. After approval of the constitution and bylaws, opera-

* Delivered before the Southwestern Surgical Congress, Houston, Texas, April 1, 1958.

tional plans were developed and established. The first meeting was held at the Shamrock Hotel Houston, Texas, September 26, 27, and 28, 1949. The *Southern Surgical Journal* was approved as the official medical journal of the society.

Since this modest beginning, organized by a handful of well qualified and highly courageous surgeons, the Southwestern Surgical Congress has grown to an organization of 1074 members, which includes the outstanding surgeons of 10 southwestern states. The scientific advances and achievements are well known to all of you. The extension of antibiotic, endocrine and vascular surgery, as well as the advances in cardiac surgery, need very little emphasis. We all are proud of the achievements that you, as individuals, and we, as a society, have made.

Since the organization of the Southwestern Surgical Congress, however, there have been many changes in the social and economic structure of organized medicine and of our country.

During the past 10 years modern medicine has become more and more deeply involved in the cross-currents of public interest, public opinion and political action. The American people have become greatly interested not only in the scientific advances of medicine but also in the methods of organizing, providing and financing hospital and medical services. On the sidelines, politicians and planners of various stripes have latched onto medical issues, some with a sincere desire to solve problems, other with both eyes on the ballot box.

During the same period of time we have seen one large, clear principle give way to a multitude of smaller, more confusing issues. This, for both doctors and the public, has made it more difficult to focus on what is right or what is wrong. It has created vague conflicts between expediency and principle, humanitarianism and common sense, lesser evils and greater evils, tradition and change.

But this situation also has provided valuable lessons—lessons which we should learn and learn well. It has demonstrated the need for constant vigilance, for medical unity and for better citizenship. Above all, it has shown that positive action is superior to negative opposition. It has disclosed a major thread to help us unravel the pros and cons of many confusing issues. That major thread is the factor of third-party interference.

To illustrate these points, let me sketch briefly some of the highlights of the past 10 years. They involve not only problems in the field of medical legislation but also third-party questions created by medicine's relations with labor, management, insurance organizations, hospitals, medical schools and other groups interested in the ways of providing medical service.

When the Southwestern Surgical Congress was being organized in late 1948 and early 1949, the medical profession was facing up to a major threat in the United States Congress. After the 1948 elections it had become apparent that the Truman Administration intended to push strongly for enactment of national compulsory health insurance. So in January, 1949, the American Medical Association, representing the entire medical profession, began its National Education Campaign to defeat that proposed legislation.

The threat was obvious; the issue was clear cut, and the battle lines were drawn. The question was whether or not the American people wanted some form of socialized medicine for the bulk of the population. Except for a very small minority, the medical profession closed ranks and presented a united front. The great majority of physicians made an effort to study the issue and cooperate in the grass roots campaign. As a result, they rallied a tremendous amount of public support for medicine's case.

From 1949 through 1952 the members of Congress could see clearly from the upsurge of public opinion that the people did not want national compulsory health insurance, or any other form of socialized medicine. The proposal never emerged from committee, and in the 1950 Congressional elections a sizeable number of its most outspoken supporters were defeated for reelection.

It is important to remember that one of our most powerful arguments against compulsory health insurance was the positive alternative of voluntary health insurance. Approximately half of the campaign effort was aimed at telling the story of the growth, availability and future promise of the voluntary plans.

Another important point to remember is the fact that even before the National Education Campaign ended in 1952, the proponents of national health legislation began to switch their strategy and tactics. Perceiving that a frontal attack could not succeed, they switched to the more subtle strategy of flank attacks and infiltration. They adopted the "piece-meal" approach and began to split up the so-called "omnibus health program" into separate bills. Even before

our campaign ended, Oscar Ewing proposed Social Security hospital benefits for people over 65, and the first efforts were made to enact the waiver of premium for disabled persons covered by the Social Security program. Other bills began to crop up on such subjects as Federal aid to medical schools, disability benefits and various types of expanded government health activities.

We were warned to beware of the changed tactics by the social planners and their political spokesmen. We were urged to remain alert and vigilant to these various side-door and back-door proposals and to push ahead on the state and local level for positive solutions of all socio-economic problems which might provide ammunition for the socializers.

Unfortunately, our profession as a whole tended to relax following the success of the fight against national compulsory health insurance. Even after the 1950 Congressional elections, before our campaign was over, many physicians got the feeling that the battle was won. However, over the past five years or more the social planners, their political spokesmen and their supporters have not been relaxing. With far too much success, they have been increasing their efforts to enact various "fringe" proposals—each one of which then serves as a precedent or stepping-off point for still another proposal. The ultimate objective is the full package of the welfare state, including a government-controlled medical system.

From 1944 through 1953 the Congress did not enact one single bill which was opposed by organized medicine, and it adopted numerous measures which received our support. Since 1953, despite the high level of prosperity and the existence of a conservative administration in Washington, we have seen the passage of more medically undesirable legislation than in any other comparable period. This was especially true of the two-year period of the last Congress, the 84th. Every year the number and variety of bills involving medicine continues to increase. So does the total number of all kinds of proposed legislation, indicating a growing reliance on Federal government to solve all problems. For example, more bills—approximately 19,000—were introduced in the last Congress than in any of the preceding 10 Congresses. Of the 19,000 bills introduced a total of 571 involved medicine to a major degree, and 25 of them were enacted into law.

The wide range of medical legislation considered by the 84th Congress included mental health, construction of medical research facilities, deferment of medical students from military service, Civil Defense medical stockpiling, vocational training for practical nurses and auxiliary hospital personnel, water pollution, treaties affecting medical licensure, the Doctor Draft Law, child health and welfare services, the commissioning of osteopaths as medical officers in the Armed Forces, Salk vaccine, the national medical library, the Jenkins-Keogh Bills, medical care for military dependents, a national morbidity survey, Social Security disability benefits, narcotic addiction, and air pollution. This is but a sampling of the varied subject matter encompassed in 571 bills.

I mention this to give you some idea of the ever-widening scope of medical legislation. The perennial bills for compulsory health insurance are still being thrown into the hoppers each year, but much more has been added. Some of it, of course, represents serious, well intentioned effort to cope with real problems. However, a great deal of the increase in proposed legislation represents deliberate ideological strategy based on the premise of "confuse and conquer." Our job in the legislative field is to distinguish the wheat from the chaff, the good from the bad, the sincere from the opportunistic.

Before we take a look at the present situation, let us review some of the legislation and other developments which medicine has viewed with concern. In 1954, for example, Congress passed the waiver of premium measure to protect the Social Security eligibility of persons who became disabled and unable to earn a living. In opposing this plan the doctors maintained that there were better alternatives not involving medical determination, and that the waiver of premium provision would simply be used as a preliminary to a disability benefits program.

We did not have to wait long for our prediction to come true. The very next year, 1955, the House of Representatives—without holding public committee hearings and with only limited debate on the House floor—jammed through a bill providing the payment of Social Security benefits at age 50 to persons adjudged to be permanently and totally disabled in relation to their ability to work. The Senate postponed consideration of this bill until 1956, which of course was to be an election year.

The American Medical Association immedi-

ately alerted the profession and initiated an educational program. Because of the public relations aspects of this problem, the educational program was directed at physicians rather than at the general public. An impressive array of well qualified witnesses presented medicine's case to the Senate Finance Committee and pointed out that the disability benefits program would be a precedent for still further expansions of the Social Security system—and they urged a thorough, impartial study of that system. The Senate Finance Committee disapproved the bill, but when it was brought up on the Senate floor it passed by the narrowest of margins—with the surprising aid of election-year votes from a few supposedly conservative senators.

So last year, and so far this year, we have seen just what was expected—a rash of new proposals to lower or eliminate the age 50 requirement for disability benefits, to establish a program of temporary disability benefits, and so on, *ad infinitum*.

The year 1956 also brought enactment of the Medicare program to provide government-financed hospital and medical care of the dependents of servicemen in the Armed Forces. The American Medical Association did not oppose that legislation because it felt that Congress had the prerogative of deciding whether or not such benefits were necessary and proper. We did emphasize, however, that any such program should try to "maximize" the amount of care in private hospitals and "minimize" the care in military facilities. After the Medicare legislation was passed, the Association established a special task force which has done effective liaison work with the Defense Department and the state medical societies to assure the best possible administration of the program.

Although many physicians are concerned over the Medicare program, it has functioned so far with efficiency and economy, and it has been generally accepted by most of the medical profession. However, it bears careful watching. Last June, for example, the A. M. A. House of Delegates condemned any kind of Medicare payments which would encourage the corporate practice of medicine; recommended that the regulations and directives be modified so that the program can be operated on an indemnity basis where desired by the individual states, and urged that Medicare regulations incorporate the Association's policy that the practice of anesthesiology, pathology, radiology and physical medicine constitute the practice of medicine. These and all other aspects of the Medicare program demand careful, constant study, for that plan may set a pattern for a similar mechanism applied to government Civil Service employees.

There is another type of problem which we face. This kind involves not new legislation, but rather the subtle, steady expansion of a program already in operation under existing laws and regulations. The major example of this is the tremendous expansion of the Veterans Administration hospital and medical care program since 1945. We now have over 22 million veterans, and each year around half a million are admitted and discharged in VA hospitals. Between 80 and 85 per cent of those discharged each year are patients with non-service connected disabilities. On any given day close to two-thirds of the VA hospital beds are occupied by non-service connected cases. Our veteran population is aging, and the cost of the VA medical program is constantly increasing.

We believe that VA hospital and medical care should be limited to veterans whose disabilities were caused or aggravated by their military service. However, as long as the present law exists preference should be given to veterans whose illness constitutes real economic hardship. This whole problem of veterans' medical care is a vital part of the basic issue of government intervention and aid *versus* private initiative. It is still unresolved, but it is going to require some vigorous educational effort by the medical profession in the near future.

So much for examples of recent issues. We might think of them as the visible portion of an iceberg—as merely the surface signs of a vast, unseen trend toward increased governmental health activities. How about the present situation? What do we face today? Despite the great current concern over earth satellites, ballistic missiles, defense spending, economic recession and scientific education, we must be alert to all possibilities. In an election year, when the members of Congress can be extremely erratic and unpredictable, we must be ready for anything.

Over 400 medical and health bills were introduced last year in the first session of the 85th Congress, and by the end of this session there undoubtedly will be a new record exceeding that

of the last Congress. Included are a large number of Social Security amendments, some of which I mentioned earlier and some of which involve the compulsory inclusion of physicians under the OASDI provisions of the act. Of all the subjects covered in this multitude of proposals, however, the most significant—the most ominous—is the provision of hospital and medical benefits for Social Security beneficiaries.

Of several bills introduced on this subject, the Forand Bill has received major attention. This bill would apply to an estimated 12 to 13 million people who are receiving or are eligible for Social Security retirement and survivorship payments. It would pay for a combined total of 120 days of hospital and nursing home care per year, and also for necessary nonelective surgery in a hospital and necessary minor or emergency surgery in a doctor's office. All of this would be provided under agreements, regulations and fee schedules arranged by the Department of Health, Education and Welfare. The Forand Bill also would increase Social Security tax rates, the amount of taxable income and cash benefits all across the board.

This dangerous legislation would impose a compulsory health insurance tax on practically all working people, while providing the benefits for just one segment of the population. It would be a big opening wedge for further expansion of benefits and coverage—and this time the expansion would involve service benefits of unpredictable cost. Since last summer the American Medical Association has had a special task force at work on this problem. More recently it created an intraprofessional campaign set-up to contain the Forand Bill in the House Ways and Means Committee if possible. But to be prepared for all eventualities, I urge all of you to acquaint yourselves with this issue and to be ready when needed—this year, next year or in 1960!

All of our problems, as I implied earlier, do not arise from legislation or third-party interference by government. We have some pretty sizeable headaches involving a wide variety of private and institutional third-parties.

For example, physicians and medical societies are frequently at odds with labor unions, industries and lay groups over the ethics and methods of their medical care plans. An outstanding illustration, with which many of you are familiar, is the current unhappiness between the medical profession and the United Mine Workers of America. The basic issue there is whether the UMWA has the right to decide arbitrarily on the qualifications and competence of physicians and hospitals, thereby limiting freedom of choice by its beneficiaries.

In some parts of the country the local medical profession has objected to the private practice of medicine by salaried faculty members in tax-supported medical schools. There also have been charges that some of the schools have been overhungry for power, prestige and publicity. After a long committee study of this problem, the A. M. A. House of Delegates decided that it is not in the public or professional interest for a third party to derive profit from payments for medical services, or to intervene in the physician-patient relationship.

Similar issues are involved in the question of the corporate practice of medicine by hospitals, as it involves financial arrangements with specialists in radiology, pathology, anesthesiology and physical medicine. Organized medicine maintains that those specialists are engaged in the practice of medicine and should receive the fees for the services they render.

All of these problems—and I have cited but a few out of many—illustrate that third-party interference is a major factor in most of medicine's policy decisions. We cannot merely condemn the existence of third parties *per se*, for in many situations they may be desirable or even necessary. What we must do, however, is to judge carefully the nature of the third-party operation. Does it interfere with the patient's freedom of choice, the physician-patient relationship, the doctor's freedom of action, the maintenance of high standards under medical direction? Is there any compulsion or exploitation affecting patients, physicians or public?

To make our policy decisions wisely, in view of the trends swirling around us in recent years, we have four major needs.

First, we must have constant and eternal vigilance. We must be alert not only to the big, obvious issues but also to the smaller, more subtle ones. We must look at both the forest and the trees.

Second, we physicians must become better citizens and take a more active part in community affairs. We must convince people that we are interested in them not just as patients, but also as friends, neighbors and fellow citizens. We must

try to get a better understanding of their opinions and problems, and try to help them solve them.

Third, we must have effective medical unity if we are to preserve the traditions and freedoms in which we believe. We can achieve such unity through our American Medical Association, which represents every branch and corner of our profession and councils, bureaus, committees and task forces studying every important issue confronting medicine today. The Association will provide leadership, guidance and practical help, but we doctors in the states and communities must provide the grass roots action which is so vital and necessary. We must work with many hands but speak with one voice.

Finally, we must translate our vigilance, our good citizenship and our unity into positive action to solve any socioeconomic problems which exist. We no longer can afford the tactics of negative opposition and passive resistance. We must pull the fuel out of the propaganda fires of the social planners, and resist all attacks upon our medical freedom. We must be eternally vigilant!

SINGLE USE DISPOSABLE DISC OXYGENATOR FOR OPEN HEART SURGERY*

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Our experience with a modified Kay-Cross disc oxygenation system in open heart surgery has been favorable except for the excessive time required to clean and assemble the equipment.

At the present time we employ the Kay-Cross stainless steel disc oxygenator using a simple syphon of one-half inch internal diameter tygon tubing to deliver blood from the inferior and superior venae cavae to the oxygenator which is placed on a table 19 inches above the floor. The oxygenated blood then passes by gravity drainage through one-half inch internal diameter tygon tubing into a "Mark-Debakey" pump and is then passed through an Edwards screen type bubble trap and filter back to the patient through one or more arterial cannulas placed in the superficial femoral artery or subclavian artery. Blood from the open heart is removed by gentle suction under a negative pressure of 20 centimeters of water through a three-eighths inch internal diameter tygon tube into a coronary suction reservoir placed at the level of the heart. The blood then passes through a screen coated with anti-foam A and in turn into the disc oxygenator by gravity.

Heparinized blood to prime the system is added through small holes in the coronary suction reservoir that accept Fenwal plastic donor blood sets. Blood can also be added during the bypass procedure to maintain the level in the oxygenator through the same route from siliconized blood bottles hung on standard I.V. poles. These bottles now function as a blood reservoir.

The system is extremely stable during the bypass procedure and requires a minimum of adjustment. The mean blood pressure and venous pressure before bypass usually is maintained easily, and blood balance has been no problem. We believe that much of our success in the use of this equipment is due to the meticulous care given to the cleaning of the equipment between runs in order that pyrogens and foreign proteins are eliminated. This has been borne out by our experience with open heart surgery in dogs with non-sterile equipment in which extensive pooling of blood and marked changes in blood pressure often occurred.

The cleaning is accomplished as follows; after each use, the equipment is immediately disassembled completely into a myriad of small parts and washed in cold water. The tygon tubing is completely discarded. After being washed in cold water, the parts are dried and all silicon grease on the oxygenator cylinder and stainless steel parts is completely removed with a solvent. The glass, stainless steel and plastic parts are then soaked in 20 per cent sodium hydroxide solution for two hours to dissolve blood proteins clinging to the surfaces after which the parts are washed thoroughly and neutralized with dilute acetic acid and again thoroughly washed. Following the final rinses and chemical tests to determine if all caustic and acid have been removed, the parts are dried in a warm oven.

Prior to the next use the stainless steel and glass parts are recoated with a thin layer of silicon grease. The oxygenator is re-assembled, wrapped and steam sterilized. The plastic parts are sterilized by soaking in aqueous zephiran until the time of use when they are thoroughly rinsed in sterile saline before assembly in a sterile field. The tygon tubing (S22-1 formulation) for each run is cut into the required lengths and is washed in distilled water, wrapped and autoclaved separately.

Two full working days for one technician are required for these procedures plus the time of one of our operating room nurses who carefully supervises the steam sterilization.

It has been obvious to us for some time that despite the great care given to the preparation of this equipment, we can not be absolutely certain that contamination is not reintroduced into the equipment during the subsequent handling after the caustic soda treatment; nor do we feel that we can afford the time of a valuable technician for breakdown, cleaning and reassembly

* This study was supported by the National Institutes of Health Grant H2618.

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of the oxygenator unit. Therefore, we have redesigned the complete system in methylmethacrylate plastic and tygon tubing so that the parts can be mass produced pyrogen-free and gas-sterilized with ethylene oxide at a low cost thus allowing the complete system to be used once and discarded. Extensive experience with methylmethacrylate plastic indicates that it is completely compatible with blood and exerts no harmful effects.

The oxygenator consists of two draw formed half shells of methylmethacrylate which fit together to form a cylinder with proper fittings in each end to receive and discharge the blood and gas (fig. 1). A deep well on the discharge end of the oxygenator insures that bubbles will not be introduced into the stream of oxygenated blood. The discs are injection molded from methylmethacrylate. The bubble trap and coronary return units consist of two half shells of methylmethacrylate plastic cemented together with a screen separating the two halves.

In use, the oxygenator units are provided in boxes completely sterile and pyrogen free with all tubing in place. A unit can be removed from a box and mounted quickly in the pump-oxygenator machine. The 13-in. unit is primed with three pints of blood and time is allowed to oxygenate the blood and to clear all bubbles from the line by slowly recirculating. The pump is then stopped and the coiled line connecting the bubble trap and the oxygenator cylinder is passed to the operating table where it is cut and connected to the venous outflow and arterial inflow lines (fig. 2). After the usual cardio-pulmonary bypass procedure, the unit can be quickly removed from the machine and discarded.

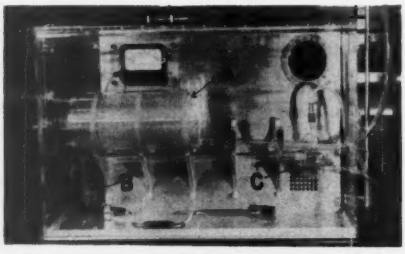

FIG. 1. *A*. Disposable plastic oxygenator. *B*. Mark-DeBakey pump. *C*. Arterial line with bubble trap and filter. Thermometer, tachometer flow meter, emergency crank and warm air ports are clearly visable.

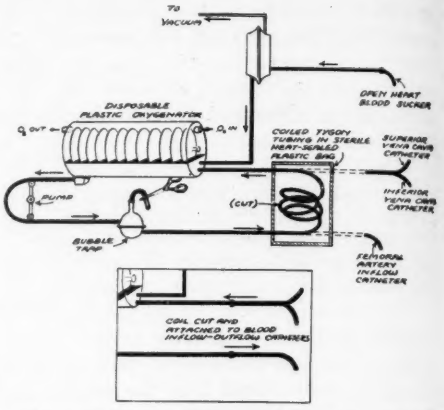

FIG. 2. Diagram of disposable oxygenator components. The coiled tygon tubing in the heat sealed plastic bag is maintained sterile during the initial oxygenation and recirculation. The tubing is then removed from the bag by the surgeon, cut and connected to the inflow and outflow catheters.

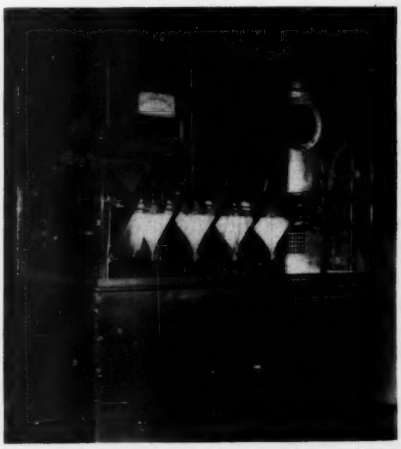

FIG. 3. Demonstrates the complete assembled unit. Note large crank which elevates and lowers unit to adjust venous pressure levels.

A simplified mechanical drive has been constructed to use with the expendable disc oxygenator (fig. 3) although there is no reason why existing machines cannot be easily and quickly modified to accommodate the units. The drive consists of a single variable speed roller pump of the Debakey type and a variable speed V pulley system to drive the rotating disc oxygenator. Both drives employ General Electric explosion proof motors and Reveo Speed changers. A Jones

mechanical tachometer indicates pump rpm and is also calibrated in blood flow in cc. per min. when the Debakey pump is set for complete occlusion. In the rare event of electric power failure during a bypass procedure, the Debakey pump and the oxygenator shaft can both be easily turned by the hand cranks provided. The metal cabinet housing the motors is covered with a plexiglass cabinet having hinged doors and easily removable ports for passing the coils of plastic tubing. The plastic cabinet is kept closed during a run, and thermostatically controlled warm air is circulated through the cabinet by an explosion proof blower and heater system. This system does does not necessarily heat the blood, but simply prevents it from cooling. The regulator can be set to add heat to the blood should this be considered necessary. The thermoregulator is an explosion proof Minneapolis-Honeywell unit. The explosion proof switches and controls are all freely accessible from the outside of the cabinet during a run.

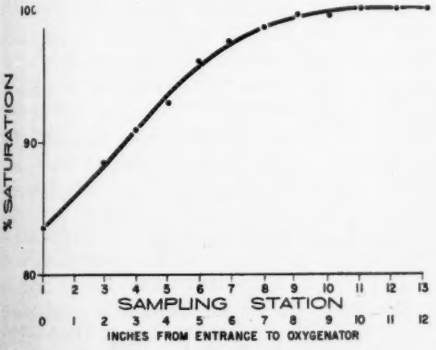

FIG. 4. Oxygen saturation of arterial blood at one-inch intervals in 13-in. oxygenator with 27-lb. dog in stable state during bypass at a blood flow of 820 cc. per min.

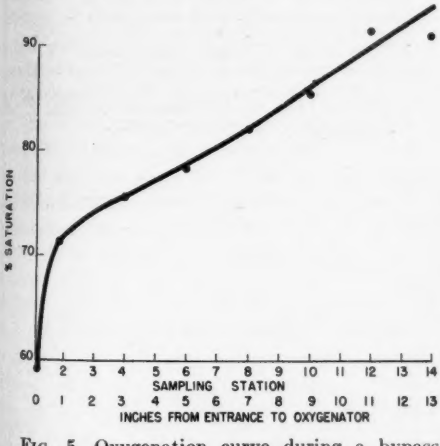

FIG. 5. Oxygenation curve during a bypass procedure using the 13-in. oxygenator at 34°C., 120 rpm, at a blood flow of 2000 cc. per min. The unit was attached to a 34-lb. and 36-lb. dog connected in parallel to provide a suitable metabolic mass at this flow.

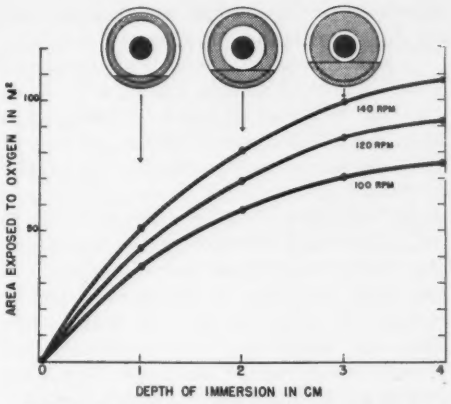

FIG. 6. Surface area of blood oxygen interface at disc speeds of 100, 120 and 140 rpm at varying immersion depths.

In order to facilitate the control of venous pressure, the entire pump-oxygenator is mounted on a Hausted adjustable level table. When the venous pressure is too low the unit can be raised to reduce the venous syphon, and an equilibrium point is easily found after which the desired venous pressure is maintained easily.

Trials in open heart surgery in the dog laboratory indicate that the expendable disc oxygenator is as effective as the Kay-Cross oxygenator and gives completely oxygenated bubble free arterialized blood. Figure 4 indicates the performance of the 13-in. oxygenator under equilibrium conditions with a 27-lb. dog at a blood flow of 820 cc. per min. The temperature was 33°C. Small holes were bored across the oxygenator just above the flange at one-inch intervals. Under conditions of equilibrium samples taken of the oxygenator blood were drawn through a long needle at each sampling station. The per cent saturation of this blood was determined photometrically, and the plot resulted in a smooth curve. It is apparent from this data that we were well within the

capacity of the intermediate size oxygenator under these experimental conditions.

In another trial to determine the maximum capacity of the 13-in. unit, a 34-lb. and a 36-lb. dog were employed in parallel to simulate a 70-lb. dog. Blood flow was 2000 cc. per min. at 34°C., and disc speed was 120 rpm. The oxygenator raised the saturation of the mixed venous blood from 59 per cent to over 90 per cent (fig. 5) in the arterialized blood issuing from the oxygenator. Under these conditions, the capacity of the small 13-in. size oxygenator was slightly exceeded. It is obvious, however, that a single 70-lb. dog would have less surface area and probably would consume less oxygen per minute.

It should be noted that the initial portion of the curve in figure 4 differs from the same portion of the curve in figure 5. This was caused by sampling at the zero station in figure 4 directly inside the entrance to the oxygenator where turbulence immediately raised the oxygen saturation of the entering venous blood. In figure 5, the sample at the zero station was obtained by needling the tygon tubing at the entrance to the oxygenator and it reflects the true mixed venous oxygen saturation level. The effect of turbulence in figure 5 is reflected in the rapid elevation of the mixed venous sample to 72 per cent at sampling station 2, together with a sharp inflection in the curve at this point.

Nine-inch, 17-inch and 22-inch expendable oxygenators are being fabricated and their capacities will be the subject of a later report.

In the use of a disc type oxygenator, the operator must always be aware that the level of the blood in the oxygenator determines the surface area of the blood exposed to oxygen, and as the level in the oxygenator drops, less and less area is exposed (fig. 6). If the oxygenator barely meets the need for a given blood flow when fully primed, every effort must be made to maintain this level during a run by adding heparinized blood from the reservoir bottles. It would be most wise in the operation of this equipment to choose a unit among the four sizes being produced having at least 50 per cent excess oxygenation capacity when fully primed to allow a safety factor for changes in blood level.

SUMMARY

An efficient expendable rotating disc oxygenator has been designed which is now being mass produced, gas-sterilized and pyrogen-free with all tubing and necessary filters for use in a blood pump-oxygenator system. The use of this equipment will greatly simplify and expedite the preparation of the pump-oxygenator for open heart surgery. The ease and ready availability of this equipment suggest application to other surgical and medical problems.

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KERATOACANTHOMA: A BENIGN CUTANEOUS TUMOR ARISING IN A SKIN GRAFT DONOR SITE*

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Keratoacanthoma (molluscum sebaceum) is a benign tumor of the skin, which recently has aroused considerable comment among dermatologists. British authors have been interested in the entity for a number of years, and lately several complete reports have appeared in the American literature. The papers of Binkley and Johnson[1] and of Epstein *et al.*[2] present the varied dermatological aspects of the tumor; while of interest to surgeons are the experiences of Burman *et al.*[3] in dealing with keratoacanthoma of the lip or "molluscum pseudocarcinomatosum," and the detailed descriptions of the histology of this lesion by McNulty and Sommers.[4]

Beginning as a small papule, the lesion of keratoacanthoma grows rapidly within a period of four to six weeks into a firm, discrete nodule 1 to 2 cm. in diameter which projects prominently above the skin surface. Resembling in many respects a fast growing squamous cell carcinoma, it may or may not ulcerate with bleeding and local infection and often shows a central crater. As a rule, the tumor develops on areas of exposure, such as the face, lips, neck, hands, and forearms, an observation which suggests that trauma or sunlight may be associated with its onset. Its appearance among workers handling petroleum products points to a more specific etiological factor. The tumor usually is single or localized, but it may develop in several areas concurrently or subsequently. In some patients as many as 200 separate lesions dot the exposed areas of skin in a generalized pattern. An unusual feature of this tumor is its ability to disappear spontaneously within a few months. Such a striking development has often been observed in instances of multiple lesions. For this reason the lesion has been variously called self-healing epithelioma or acanthoma, tumor-like keratosis of Poth, and benign epithelioma.

Its close resemblance to squamous cell carcinoma has prompted extensive discussion among clinicians and pathologists as to whether or not it is a true malignant neoplasm. Histologically the growth of the epidermal elements is exuberant, yet it follows an over-all orderly pattern. The cells actively proliferate but not into atypical forms as a rule. Pools of keratinized debris occupy much of the central portion of the lesion, often creating a central depression. The pathologist may have difficulty in determining whether the lesion is keratoacanthoma or low grade squamous cell carcinoma from the histological picture. Once acquainted with the entity, he relies heavily on the history of its unique clinical attributes before finally deciding upon that diagnosis. Even then, he is apt to feel uneasy unless the lesion is handled as though it were a neoplasm of low grade malignancy. Some dermatologists, on the other hand, have advocated expectant treatment of keratoacanthoma, relying on its clinical proclivity to involute into a hard, white scar. Such a course is probably justifiable in treating multiple tumors once the histological pattern has been established, but it is not warranted in the case of single lesions that simulate so closely squamous cell carcinoma.

The following case report illustrates many of the peculiar and arresting features of keratoacanthoma, including some not previously described, as far as the author can determine.

CASE REPORT

An 82-year-old white retired caretaker, of light complexion, presented with a 4-cm. fixed, firm mass overlying the left sternocleidomastoid muscle and apparently arising primarily in the skin. The lesion was superficially ulcerated and adherent to the underlying muscle. It had been slowly growing over a period of several years despite intermittent x-ray therapy. The diagnosis was basal cell carcinoma. In addition to the main lesion, scattered pigmented and senile keratoses covered the face, neck, and upper trunk. In other respects the patient's health and condition were good.

On 10/17/57 under general anesthesia the tumor mass on the neck was excised with a margin of

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FIG. 1. The entire specimen, measuring 17 mm. in diameter, is pictured as resected from the donor site on the thigh. Florid overgrowth of the epithelial elements with acanthosis and hyperkeratosis is striking. The pattern is regular without sign of invasiveness. Diagnosis: keratoacanthoma. × 8.

FIG. 2. Cellular detail of the lesion shown in figure 1. Despite active epithelial growth the cells retain normal characteristics and do not show atypism as does low grade squamous cell carcinoma. × 160.

normal tissue including muscle and skin. The defect, approximately 6 cm. in diameter, was covered with a thin, split-thickness skin graft taken from the anterior surface of the left thigh with an electric Brown dermatome. The donor site, previously healthy and smooth, was prepared by shaving and applying mineral oil. The donor site on the thigh healed promptly within 12 days, after being dressed with plastic surfaced Telfa gauze. Pathological diagnosis of the resected tumor was basal cell carcinoma with some squamous cell elements.

A small, firm, slightly tender, 1 mm.-papule was first noted in the center of the healed donor site 22 days after the operation and within 1 week it grew to 4 mm. The papule was incised in the belief that it represented a crusted pustule, but it proved to be solid. Over the next 4 weeks it grew rapidly and steadily into a single, firm, nonulcerated, slightly umbilicated nodule about 15 mm. in diameter and raised prominently above the skin. The patient complained about its being constantly tender and sensitive to the friction of his trousers. Its rapid growth and clinical features suggested strongly a malignant neoplasm of the skin arising in a donor site, possibly a tumor transplanted at the time of surgical excision. Meanwhile the grafted area where the primary tumor of the neck was excised remained well healed without evidence of recurrence.

On 12/4/57 the nodule in the donor site was excised under local anesthesia. The incision healed promptly, and there has been no further tumor formation. The initial pathological diagnosis of the resected tumor from the donor site (figs. 1 and 2) was squamous cell carcinoma, grade I. Upon review of the histological sections in the Department of Pathology of the University of Cincinnati, the diagnosis of keratoacanthoma was established.

DISCUSSION

The entity of keratoacanthoma is dramatic because of its startling resemblance to squamous cell carcinoma and its rapid growth. A further peculiarity is its tendency to spontaneous involution after several months if no therapy is begun. The lesion may be multiple and may recur after inadequate excision, but it has not been known to invade lymphatic tissue or metastasize in the sense of a carcinoma. The microscopic characteristics suggest carcinoma, but the over-all pattern and the individual cells remain orderly and conservative. Most authorities, while maintaining that keratoacanthoma is not a malignant neoplasm, warn that histologically it often cannot be easily distinguished from low grade squamous cell carcinoma, and that the distinction rests on the special clinical features of the lesion rather than its microscopic appearance.

The surgical therapy of single lesions should be planned as though intended for a squamous cell carcinoma, an approach which results in cure and obtains the entire growth for histological study. However, in areas about the face, radical excision of this benign growth is unnecessary, and simple excision suffices. X-ray therapy, cautery, and conservative observation have been used, but with such modes of treatment there is no certainty about the diagnosis. A correct diagnosis of keratoacanthoma has obvious importance for determining accurate therapy, prognosis, and useful information in industrial cases.

To the author's knowledge, the case here reported is the first to arise in a skin graft donor site. It is interesting to speculate whether the petroleum product, mineral oil, or the trauma of removing the graft initiated its rapid growth only three weeks later. Throughout its active development the lesion was closely observed with some concern by both the patient and surgeon, and when its identity was established, both felt considerable relief.

The entity is not rare, and should be better known to surgeons and surgical pathologists. Its importance lies more in its being confused with squamous cell carcinoma than in any inherent dangerous qualities of its own. Many examples have probably been catalogued as low grade carcinomas of the skin, thereby falsely influencing the cure rates of that cancer. Awareness of the unusual features of keratoacanthoma should readily identify it to surgeon and pathologist.

SUMMARY

Keratoacanthoma is a benign, self limiting tumor of the skin that resembles closely squamous cell carcinoma of the skin both grossly and microscopically. Rapid growth and spontaneous involution characterize the lesion. Usually single, but occasionally multiple, it appears chiefly on exposed areas and has been noted particularly on patients working with petroleum products. A case is reported in which a keratoacanthoma grew rapidly in a healing donor site of a split-thickness skin graft. An accurate diagnosis of this tumor is important for prognostic as well as therapeutic reasons. Undoubtedly in the past many instances

have been mistakenly labeled as squamous cell carcinoma.

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VENTRICULAR FIBRILLATION DURING HYPOTHERMIA: FURTHER OBSERVATIONS*

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INTRODUCTION

Hypothermia and extracorporeal pump-oxygenation each permit manipulation within the opened chambers of the heart. The former protects such vital centers as the central nervous system and heart from hypoxia during periods of circulatory occlusion by lowering their metabolic rates thermally,[21, 20] whereas the latter provides adequate volumes of oxygenated blood to normothermic tissues during periods of heart-lung exclusion.

Currently, hypothermia is employed in operations within the great vessels and atria, but because of the ease with which ventricular fibrillation occurs during manipulation of the cold ventricles, extracorporeal pump-oxygenation is usually used during open cardiotomy of these lower chambers of the heart.

Since the reports of Bigelow[2] in 1950 concerning oxygen consumption at low body temperatures and its possible relation to cardiac surgery, and since the report of the first successful closure of an interatrial septal defect under direct vision by Lewis and Taufic,[14] hypothermia has proved to be a great asset in operations upon the heart and great vessels, exclusive of the cardiac ventricles.[24, 25, 26, 27]

Clinically, in our hands, total body cooling, as reflected by the rectal temperature, is usually not carried below 30°C. The duration of circulatory occlusion at this temperature is confined to 6 min. because of the increased frequency with which cardiac arrhythmias and postoperative bleeding tendencies occur beyond these limits of time and temperature.[23]

The canine brain will withstand temperatures of 11° to 13°C.[19] However, as the heart is cooled below 30°C. it becomes increasingly susceptible to first, ventricular fibrillation, and, at lower temperatures, to standstill. It is mainly the propensity to develop ventricular fibrillation which limits the temperature, and thus the duration, of intracardiac manipulation. Until this arrhythmia is controlled, lower ranges of hypothermia cannot be entered safely, and longer periods of circulatory occlusion may not be anticipated.

In 1954, Montgomery *et al.*,[18] in our laboratory, demonstrated that neostigmine was effective in preventing ventricular fibrillation in hypothermic dogs when it was administered by coronary perfusion; *i.e.*, injected into the root of the cross-clamped aorta. In these animals, the chest was entered through a right thoracotomy, and circulation was arrested during the performance of a right ventriculotomy by occlusion of the inferior and superior vena cavae at their entrance into the right atrium; *i.e.*, "inflow occlusion."

It is assumed that the virtue of neostigmine rests in its ability to inhibit cholinesterase and thus prolong the action of acetycholine within the heart, because the resultant bradycardia and decreased incidence of ventricular fibrillation in the hypothermic heart is similar to that obtained from the coronary perfusion of acetylcholine itself.[13, 23]

The synthesis of acetylcholine is contingent upon the formation of ATP (adenosine-triphosphate), which is produced more efficiently in the aerobic (Krebs cycle) metabolism of glucose than in the anaerobic phase, (Meyerhoff scheme). Therefore, acetylcholine is also synthesized most efficiently in an aerobic environment, and neostigmine would be expected to be most effective under similar conditions. Such circumstances prevail within the hypothermic myocardium when the general circulation is arrested for short periods by "inflow occlusion" alone, due to slow but significant perfusion of the coronary arteries with oxygenated blood from the aorta.

In addition to an "inflow occlusion" during clinical cardiotomy at this medical center, a non-crushing clamp is placed through the transverse pericardial sinus and across the roots of the aorta and pulmonary artery; *i.e.*, "outflow occlusion."

* Supported by a Grant from the American Heart Association and by a Contract with the United States Army, Office of the Surgeon General, DA-49-007-MD-572.

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This serves to obstruct the coronary ostia and protect them from air embolization. However, it also abruptly stops the coronary perfusion of arterial blood, diminishes the production of ATP and acetylcholine, and renders neostigmine less effective as an antifibrillatory agent.[1, 9] Therefore, a compound was sought which would act like acetylcholine itself, would not be rapidly metabolized, and would be effective in such a relatively anaerobic myocardium. In 1955, Baer *et al.*[1] from this laboratory, reported the use of Urecholine (bethanecol chloride, Merck) in dogs undergoing right ventriculotomy and unilateral thoracotomy, at temperature ranges of 25° to 27°C. This agent was found to meet the desired criteria and to prevent ventricular fibrillation during periods of inflow-outflow occlusion of 10 to 15 min. The suggested dosage was 0.006 to 0.010 mg. per kg. of body weight, to be given by coronary perfusion. In the event that bradycardia persisted after release of circulatory occlusion, atropine was suggested as an antidote. Most of the experiments were acute, so that the toxicity of Urecholine could not be adequately assessed.

The original objects of the current investigation were to verify the dosage of Urecholine, to determine its toxic limit, to evaluate atropine as an antidote, and to express its dosage in relation to that of Urecholine.

METHODS

Mongrel dogs were used, varying in weight from 6.4 to 16.3 kg. The ratio of males to females was 4:3. All the animals were chosen at random from a large "isolation run" where they had been kept following their arrival from the municipal dog pound. The only selection involved was an attempt to exclude any animals which were obviously ill or in the extremes of age. None received any special preoperative diet or medication. No preanesthetic medication was employed. Sodium pentobarbital was administered intravenously as an anesthetic, although there is some controversy regarding its role in ventricular fibrillation in hypothermia.[7, 17]

The dosage was adjusted to cause elimination of the lateral canthal reflex with preservation of the medial, which required approximately 30 mg. per kg. of body weight. An intravenous infusion of either 0.9 per cent NaCl or Ringer's solution was begun, and maintained at a slow rate throughout the cooling, operative, and early postoperative periods. The animals thus received 100 to 200 ml. of the solution. The thorax was clipped and shaved. The trachea was intubated snugly, but not occlusively. The animal was placed in an ice-water bath which covered the body and proximal portion of each limb. The paws were carefully protected from immersion to avoid peripheral neuritis. Positive pressure respiration with 100 per cent oxygen was begun shortly after immersion at a rate of 40 to 60 per min. to prevent hypoxia or hypercapnia and to maintain a stable, slightly alkaline[29, 3, 28, 11] blood pH. When a rectal temperature of 29°C. was attained, the animal was removed from the ice-water and placed upon the operating table. Thereafter, the rectal temperature declined an additional 3° to 5°C., so that all of the following investigations occurred at a rectal temperature of 24° to 26°C.

Each of the operated animals underwent a bilateral trans-sternal thoracotomy through the fourth intercostal space. This approach was selected to afford optimal exposure of the heart and great vessels and, more particularly, to simulate the clinical situation. Cotton tapes were placed about the inferior and superior venae cavae at their junction with the right atrium, the latter tape being so placed as to afford occlusion of both the superior vena cava and the azygos vein.

The pericardium was opened sufficiently to permit performance of a right ventriculotomy. To achieve "inflow occlusion," the cotton tapes were tightened. After allowing 30 sec. for cardiac emptying, the aorta was cross-clamped 0.5 to 1.0 cm. distal to the coronary ostia. Injections into the root of the aorta were made and 30 sec. allowed for myocardial perfusion, which was achieved by cardiac action itself or intermittent manual compression of the heart. The aortic clamp was then removed and a straight Harken's clamp was introduced through the transverse pericardial sinus across the root of the aorta and pulmonary artery to achieve "outflow occlusion." A 2 to 3-cm. right ventriculotomy was made between forceps and stay sutures. The interventricular septum and pulmonary and tricuspid valve orifices were firmly palpated. The ventricle was closed with a continuous 5-0 or 3-0 silk suture, the speed of closure being adjusted so that the myocardium was stimulated intermittently throughout the period of occlusion. Only occa-

sionally was an attempt made to exclude air from the right ventricle following ventriculotomy.

Outflow occlusion was released, followed by restoration of the superior vena caval flow. Atropine sulfate, when indicated, was administered by coronary perfusion to increase the cardiac rate. Inferior vena caval flow was reconstituted after adequate myocardial contractions were present—about 2 to 3 min. after loosening the superior vena caval tape.

During all but the first few months of this investigation, a routine of thoracotomy closure and early postoperative care was employed which yielded an operative mortality of less than 5 per cent and which, therefore, is outlined below:

1. Closure of the pericardium to afford support to the heart and prevent obstruction of vena caval return through torsion.
2. Placement of intrathoracic catheters bilaterally with under-water negative pressure drainage for 5 to 10 hr. postoperatively.
3. Careful closure of the thorax:
   (a) Approximation of the sternum with two no. 28 stainless steel parasternal wires.
   (b) Approximation of the rib cage with a single no. 28 stainless steel paracostal wire, bilaterally.
   (c) Closure of the intercostal bundle and pectoral muscles as separate layers with continuous 2-0 chromic catgut.
   (d) Closure of the superficial fascia with continuous 3-0 chromic catgut.
   (e) Approximation of the skin edges with continuous 3-0 black silk.
4. Administration of 10 ml. per kg. of blood or a plasma volume expander immediately postoperatively.
5. Rapid (over the course of 1 to 1½ hr.) rewarming in a water bath at 40° to 45°C. Spontaneous respirations were allowed to resume when the animal reached a rectal temperature of 32° to 36°C.
6. Intramuscular administration of Metrazol, 100 mg. (1 ampoule), per kg., to encourage deep breathing in the early postoperative period, and early shivering during the rewarming phase. Convulsions did not occur.
7. Routine bronchoscopy on the morning of the first postoperative day of all animals weighing 10 kg. or less, and of any other animal which developed a cough or evidenced respiratory difficulty in the early postoperative period.
8. Intramuscular penicillin and streptomycin on the day of operation and on each of the first three postoperative days.

If ventricular fibrillation occurred at any time during the cardiotomy, it was allowed to persist throughout the predetermined period of occlusion. Electrical defibrillation was attempted in accordance with a protocol which stresses assurance of good myocardial tone and oxygenation prior to application of a short high-voltage shock with moistened, padded, electrodes.[12]

In all phases of this investigation, "survival" was defined as "alive and well" seven days postoperatively.

RESULTS AND DISCUSSION

I. *Prevention of ventricular fibrillation.* In an attempt to verify the favorable results obtained by Baer *et al.*[1] with Urecholine as an inhibitor of ventricular fibrillation, 17 dogs received this agent by coronary perfusion prior to right ventriculotomy from July 6, 1955 to August 15, 1955. The dosage was varied from 0.001 mg. per kg. to 0.750 mg. per kg., and was adjusted to a volume of 1.0 ml. with 0.9 per cent NaCl. Of these animals, 16 developed ventricular fibrillation at some point during an 8 to 10 min. occlusion, usually when the epicardium was grasped with forceps or when the myocardium was incised. Fibrillation occurred in spite of the fact that doses of 0.032 mg. per kg. or greater caused either complete cardiac standstill or a severe bradycardia, usually the former; implying a preponderant parasympathomimetic stimulation. Displacement of autonomic balance in favor of the parasympathomimetic by sympathectomy, vagal stimulation, or by administering sympatholytic or parasympathomimetic agents has been shown to inhibit ventricular fibrillation under varying conditions.[18, 1, 22, 4, 10, 5, 13, 8]

Quantities of Urecholine in excess of that required to achieve cardiac standstill also appeared to have no antifibrillatory effect.

Following the release of circulatory occlusion, intracoronary atropine, 0.100 mg. to 0.600 mg. (total, not per kg. of body weight), always successfully counteracted the depressant effect of Urecholine. A more precise dosage of atropine was found to be one quarter of the corresponding dose of Urecholine, *i.e.*, mg. of atropine = mg. of

Urecholine/4. Ventricular fibrillation did not follow the coronary perfusion of atropine sulfate.

This high incidence of fibrillation was discouraging. Three factors were considered to be relevant: the inexperience of the new personnel who arrived in the laboratory on July 1, 1955; the possible liberation of substantially more epinephrine into the general circulation by the added trauma of the bilateral trans-sternal thoracotomy, circulatory epinephrine itself being a powerful stimulus to ventricular fibrillation in the hypothermic heart;[1] or a seasonal difference in the irritability of the canine myocardium, permitting ventricular fibrillation to occur more easily in the warm summer months—an impression held by us and others.[6]

The factor of technique seemed adequately eliminated by having permanent members of the laboratory staff repeat the experiment on several occasions with the same discouraging results.

The measurement of circulating epinephrine in dogs undergoing a bilateral thoracotomy as contrasted to those with a unilateral thoracotomy is planned.

A series of experiments was started to evaluate objectively any seasonal variation in myocardial irritability. Further investigation of Urecholine was postponed until the winter months.

From November 11, 1955 to January 3, 1956, five animals underwent right ventriculotomy after the coronary perfusion of Urecholine, 0.050 mg. per kg., adjusted to a volume of 1.0 ml. with 0.9 per cent NaCl. This dosage was selected because it invariably caused a marked bradycardia or cardiac standstill. Such relative cardiac inactivity was considered to be desirable because: it indicated a maximal inhibition of normal conduction and thus implied a maximal parasympathomimetic stimulation; it provided a quiet field for intracardiac manipulation; it probably lowered myocardial metabolism, and thus served as some protection to the heart during the occlusion period; it was a state easily reversed by the coronary perfusion of atropine sulfate following the release of occlusion. Four of these five dogs fibrillated.

During a comparable period, November 17, 1955 to January 17, 1956, five additional animals were similarly treated. However, the volume of perfusate was adjusted to 3.0 ml. with 0.9 per cent NaCl, rather than to 1.0 ml., to achieve more complete perfusion to the myocardium. Three of these animals suffered ventricular fibrillation.

Apparently, inhibition of the normal conduction pathways within the canine myocardium was insufficient to prevent ventricular fibrillation under our experimental conditions. That the myocardium retained a high degree of irritability was inferred from the ease with which ventricular fibrillation developed and from the vigor of the isolated contractions obtained when the arrested myocardium was manipulated. A suitable myocardial depressant was sought.

Preliminary studies were made combining Urecholine with Pronestyl* (procaine amide, Squibb), an agent known to offer protection against ventricular arrhythmias.[15] Immediately thereafter, we received a quantity of Ambonestyl† for investigative purposes and concomitantly Clark and Lanzoni[5, 13] reported upon their experiences with this agent. Ambonestyl was introduced into our experimental regimen in the place of Pronestyl. The two agents have been observed to possess equivalent anti-arrhythmic activity; however, the side effects of Ambonestyl are considerably less.[5]

It became evident that success depended upon the intracoronary perfusion of Ambonestyl, 19 to 15 mg. per kg., 15 to 30 sec. prior to the introduction of Urecholine 0.05 to 0.10 mg. per kg. Fourteen dogs so treated underwent right ventriculotomy during occlusions of 8.5 to 13 min. from September 22, 1955 to August 8, 1956. Ventricular fibrillation did not occur and all of the animals survived. A strong regular sinus rhythm was easily achieved with atropine sulfate after the release of occlusion. It is presumed that the introduction of Ambonestyl must precede that of Urecholine to allow adequate myocardial perfusion of the former before effecting cardiac arrest.

Recently, Covino and Hegnauer[8] have reported success with *intravenous* Ambonestyl alone in the unilaterally thoracotomized hypothermic animal. They noted that this agent maintained the same low level of cardiac excitability at 23°C. and 30°C. as was present at the control temperature of 38°C.

To assess the value of intracoronary Ambonestyl alone, it was administered in doses of 10 to 15 mg. per kg. prior to cardiotomy in five animals, all of whom fibrillated at some time during the procedure—three during ventriculotomy and two post-occlusion during attempts to improve

* In the first two animals only
† E. R. Squibb & Sons, New York

ineffective myocardial contractions. Myocarcial tone became poor following the coronary perfusion of Ambonestyl. It was considered that this may have been the result of poor cardiac filling and coronary perfusion secondary to hypotension caused by the introduction of this agent into the general circulation upon release of occlusion. The production of hypotension as one of the side effects of intravenous Pronestyl has been described.[5] It was felt advisable to assess the hypotensive effect of Ambonestyl in the dosage and temperature range employed in this investigation.

Five animals were cooled as described previously and given 15 mg. per kg. of Ambonestyl in 25 ml. of 0.9 per cent NaCl, intravenously, over a 2 to 3 min. period to duplicate the conditions prevailing if the total intracoronary dose was indeed washed into the general circulation following release of occlusion. In four of these dogs, an immediate transitory hypotension of from 5 to 20 mm. Hg. occurred, accompanied by a decrease in pulse rate of from 2 to 10 beats per min. In that animal in which the blood pressure dropped 20 mm. Hg., there was a coincident rise in pulse rate of five beats per minute. It is inferred that this mild hypotension may be a contributing—but is not a precipitating—factor in poor post-occlusion myocardial tone, and that the primary factors are the combined depressant effects of myocardial hypoxia and Ambonestyl, itself.

From November 14, 1955 to August 29, 1956, 16 control animals underwent right ventriculotomy without pharmacologic protection. Ventricular fibrillation developed in 13 of these.

The occurrence of ventricular fibrillation following the combination of Ambonestyl and Urecholine is compared with that following either agent alone (tables 1 and 2) and with the control group (table 3). The success of the combined agents is statistically significant in each circumstance; *i.e.*, $p < 0.01$.

II. *Seasonal differences in canine myocardial irritability.* During a "summer" period from August 26, 1955 to September 8, 1955, 7 animals, cooled as previously described, underwent bilateral high dorsal sympathectomy and stellate ganglionectomy, plus bilateral cervical vagotomy. During an 8- to 9-min. inflow-outflow occlusion, a right ventriculotomy was made without pharmacologic protection while the heart was held in arrest by continuous stimulation of the distal cut vagi. Six of these hearts developed ventricular fibrillation, three while the ventricles were in arrest and three after "vagal escape" with the development of a slow ideo-ventricular beat. The average time of onset of fibrillation after the beginning of the ventriculotomy was 1 min., ranging from 0 to 3 min.

In a similar group of six animals operated during the "winter" period from January 25, 1956 to March 2, 1956, two developed ventricular fibrillation. In both, fibrillation occurred after

TABLE 1

Protection of the canine heart against ventricular fibrillation during hypothermia and right ventriculotomy by Urecholine and Urecholine plus Ambonestyl

Group	Fibrillation	No Fibrillation	Total
1. Urecholine (0.05 mg./kg. in 1.0 ml.)	4	1	5
2. Urecholine (0.05 mg./kg. in 3.0 ml.)	3	2	5
3. Urecholine (0.05–0.10 mg./kg.) plus Ambonestyl (10–15 mg./kg.)	0	14	14

TABLE 2

Protection of the canine heart against ventricular fibrillation during hypothermia and right ventriculotomy by Urecholine plus Ambonestyl and Ambonestyl alone

Group	Fibrillation	No Fibrillation	Total
1. Urecholine (0.05–0.10 mg./kg.) plus Ambonestyl (10–15 mg./kg.)	0	14	14
2. Ambonestyl (10–15 mg./kg.)	5	0	5

TABLE 3

Protection of the canine heart against ventricular fibrillation with and without ambonestyl plus Urecholine

Group	Fibrillation	No Fibrillation	Total
1. Control	13	3	16
2. Ambonestyl (10–15 mg./kg.) plus Urecholine (0.05–0.10 mg./kg.)	0	14	14

TABLE 4

Right ventriculotomy during cardiac sympathectomy and vagal stimulation

Group	Ventricular Fibrillation		Total
	Yes	No	
Summer (8/26/55–9/8/55)	6	1	7
Winter (1/25/56–3/2/56)	2	4	6

TABLE 5

Ventricular fibrillation in the sympathectomized heart during right ventriculotomy and continuous vagal stimulation

Group	Ventricular Fibrillation		
	During arrest	After escape	Average time after occlusion
Summer (8/26/55–9/8/55)	3	3	1 min. (0″–3″)
Winter (1/25/56–3/2/56)	0	2	7 min. (6.5″–7.5″)

"vagal escape," the average time after ventriculotomy being 7 min. (see tables 4 and 5).

The statistical significance of the difference in occurrence of ventricular fibrillation in the two groups is insecure inasmuch as $p = 0.05$. This may well be a reflection of the small number of animals involved.

CONCLUSIONS

Ventricular fibrillation during hypothermia may involve two of the functions of the myocardium: conduction and excitability. In some preparations, control of the normal conduction pathways is sufficient to offer protection against ventricular fibrillation during right ventriculotomy. However, control of this single element with intracoronary Urecholine in dosages of 0.001 to 0.750 mg. per kg. and in volumes of 1.0 to 3.0 ml. is inadequate under those conditions which parallel the clinical situation; *i.e.*, bilateral trans-sternal thoracotomy, with cessation of coronary blood flow. It is suggested that an increase in circulating epinephrine associated with the more extensive procedure may be a contributing factor.

A reduction in myocardial excitability alone, with intracoronary Ambonestyl 10 to 15 mg. per kg. is similarly ineffective as an inhibitor of ventricular fibrillation. Simultaneous suppression of both myocardial excitability and conduction with Ambonestyl and Urecholine prevents ventricular fibrillation in summer and winter, during those periods of occlusion which have been shown to be safe at the temperature range of 24° to 26°C.; *i.e.*, up to 12 minutes.[16] The cardiac arrest obtained with Urecholine is readily reversed with intracoronary atropine sulfate, 0.100 to 0.600 mg., or a dosage in milligrams equal to one-fourth that of Urecholine. The cardiac effects of Urecholine do not appear to increase with increasing doses of this agent, once arrest has occurred.

The canine myocardium seems more irritable in the summer than in the winter. No reason for this difference is offered. However, it is suggested that reports of investigations on ventricular fibrillation in the canine heart bear the dates during which such investigations were undertaken.

SUMMARY

A technique of inducing hypothermia in the dog and performing a right ventriculotomy under conditions of inflow-outflow cardiac occlusion is described. This technique parallels that used in performing cardiotomy in human patients, and as such is recommended for investigative purposes.

A method of arresting the heart and preventing ventricular fibrillation under the above conditions is described, using an intracoronary perfusion of Ambonestyl and Urecholine. Atropine sulfate is shown to be an effective antidote. Evidence is offered which suggests that a seasonal variation in success might be expected.

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A CLINICAL AND LABORATORY COMPARISON OF THE EFFECTS OF COMMONLY USED VASODILATOR DRUGS ON ARTERIAL DISEASES*

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For many years the concepts concerning vasodilators have been the subject of much debate. It is, however, an established fact that certain drugs act on specific vessel groups, producing dilation and apparent increase of blood flow. Foremost in this group is nitroglycerin, which acts primarily on the coronary vessels. Also, nicotinic acid and its analogues are known to produce cutaneous capillary dilation and flushes, primarily in the upper half of the body. Carbon dioxide is perhaps the most potent vasodilator of the cerebral circulation, but elsewhere in the body it produces variable effects on vasomotor tone. These are a few known examples of drugs that have, for poorly understood reasons, narrowed selectivity of action in certain systems of arteries.

Vasodilation may occur in response to stimulation of three different mechanisms in the regulation of vasomotor tones. The process of vasodilation resulting from the direct action of drugs on the vessel musculature can be readily demonstrated by histamine contact with vessels isolated by the quartz-rod technique of Knisely. Sympathetic nerve blocking drugs such as dibenamine hydrochloride, also produce systemic vasodilation by interruption of the ganglionic synapse obviating the constrictive influence of sympathetic fibers. The adrenolytic drugs, notably Regitine, act on arteries indirectly by blocking or antagonizing the effects of circulating epinephrine and nor-epinephrine. There are many other types of drugs which may, for example, combat vasopressor agents and tranquilize the central nervous system, thus producing ultimate diminution of vasomotor tone. These drugs are not properly classified as vasodilators, but they, by preventing vasospasm, increase the peripheral blood flow.

It is well known that generalized vasodilation lowers the blood pressure by decreasing the peripheral resistance. The natural questions concerning all peripheral vasodilators are as follows: (1) If there is vasodilation of all peripheral arteries, why is the blood pressure not affected? (2) If the total flow of blood is increased to all extremities, where is the source of this extra blood? (3) Are the effects from the drugs caused by increased cardiac output alone? (4) Do these drugs increase the flow through the arteries of the extremities and, at the same time, decrease the flow in the visceral, kidney, cardiac, or other systems of arteries?

Realizing the inconsistencies of not only the drug-artery response, but also the reports concerning the use of these drugs, a study was initiated in an effort to categorize the clinical and laboratory effects of three commonly used and one newly released so-called peripheral vasodilators. It was recognized that all the foregoing questions could not be answered in a patient study alone. The project, therefore, was divided into two parts. One was to deal with some of the measurable human responses to the drugs and the other was to be carried out in the animal laboratory. This presentation deals only with observations made on human patients.

METHODS

Arlidin, Ilidar, Priscoline, and Drug 5029 were used in this investigation and are identified briefly below:

Arlidin (nylidrin hydrochloride) is a synthetic compound belonging to the epinephrine—ephedrine group. Its primary sympathomimetic actions[1, 2] are to dilate the small vessels in skeletal muscle and to increase cardiac output. Arlidin was administered in the dosage of 5 mg. subcutaneously in three patients and 5 mg. in 250 cc. saline intravenously over a 30-min. period of time in remaining patients. In that this drug was given subcutaneously rather than intravenously in 3 patients, the results may have been inconsistent.

Ilidar hydrochloride (azapetine) is a dibenza-

* Supported by the John A. Hartford Foundation, Inc.

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sepine adrenergic blocking drug. It has been described as being adrenolytic and sympatholytic,[3] having direct vasodilating effect on arteries,[4] and having an epinephrine reversal property.[5] This drug was administered in dosage of 0.8 mg./kg. in 250 cc. of normal saline solution with the drip regulated so as to administer the entire solution over a period of 30 min.

Priscoline (benzyl-imidazoline hydrochloride) is related to both sympathomimetic amines and histamines. This drug is reported by Alilquist[6] to act synergistically with epinephrine on the animal heart and to increase cardiac output. In addition, Grimson[7] described it as being an adrenolytic and sympatholytic agent. It was administered in doses of 75 mg. in 250 cc. saline solution, regulated so as to be administered over a period of 30 min.

Drug 5029 (phenoxyisopropylnorsuprifen) is a product imported from Europe. Most of the published reports are from Germany and indicate this drug's properties and structure to be similar to nylidrin hydrochloride. It dilates the blood vessels of both skin and muscles and relieves vasospasm, according to Brucke,[8] who further reports that in the isolated heart-lung preparation of the dog, there is no increase in cardiac output. Hertting,[9] on the other hand, states the cardiac output is increased by Drug 5029 in the unanesthetized dog. It was administered in a normal saline solution of 0.5 mg. per kg. in 7 patients and 1 mg. per kg. in 7 patients. The latter dosage appears to be excessive and produces more side reactions.

These studies were made using the methods previously described in detail.[10,11] In essence, 15 patients with peripheral vascular diseases were studied before, during, and after separate infusion of the four drugs previously mentioned. All studies were carried out in a constant-temperature-humidity environment at temperature of 70°F. and a humidity of 65 per cent. These patients were brought into the constant temperature room at metabolic rest and a period of adjustment for about one hour was allowed in each case prior to beginning the study. Controls were obtained and the patient was requested to describe any unusual sensations which might occur during or after the infusion. The following examinations were recorded:

(a) *Direct evaluation.* Observations of the vessels of the bulbar conjunctiva were carried out, using a specially mounted Leitz binocular dissecting microscope which provided good resolution up to 96 diameters. The criteria utilized in grading the degree of anatomical and physiological abnormality have been described previously, but are altered as follows:

For purposes of quantitative recording, the conjunctival vessel response has been classified as *None, Minimal, Moderate, Marked, Maximal.*

None—Essentially no change.

Minimal—Slight increase in flow; generally slight increase in number of visible capillaries and arterioles, but no change in moderate sized vessel caliber (50 microns).

Moderate—Greater increase in flow; significant increase in number of visible small vessels; frequently accompanied by venous hyperemia; slight degree of increase in caliber of moderate size (50-micron) vessels.

Marked—Obvious increase in number of vessels of capillary and arteriole beds, with appreciable increase in velocity of flow, and gross vasodilation.

Maximal—Intense hyperemia with dilation of all vessels, marked increase in flow velocity, venous engorgements, numerous new channels visible.

The presence or absence of arterial aneurysms or venous sacculations and individual plugged vessels was noted as well as the degree of vascularity of the conjunctival surface. Vascularity was considered a result of vessel size and number. Changes in rate of flow as observed through the microscope were recorded and the incidence of evidence of leaking plasma through separate vessels was indicated. Following these observations, occasional photographs were taken of the microscopic fields. The films were taken primarily to record gross pathologic changes and the degree of alterations in vascularity of the field.

Photographs (fig. 1) were taken with a 35-mm. Alpa Alnea camera with a Heiland Strobonar light attachment. PX 135 film was used. Attemps were made to record the same field of view in photographs of the same patients, but this was not always successful.

(b) *Indirect evaluation.* Plethysmographic recording (fig. 1) of the digital pulse volume was obtained by means of metal or plastic cups fitted over the digit and connected by plastic tubing to a direct-writing plethysmograph. (A Grass Model III-D electronencephalograph, a balance modulator unit, and a PT-5 transducer equipped

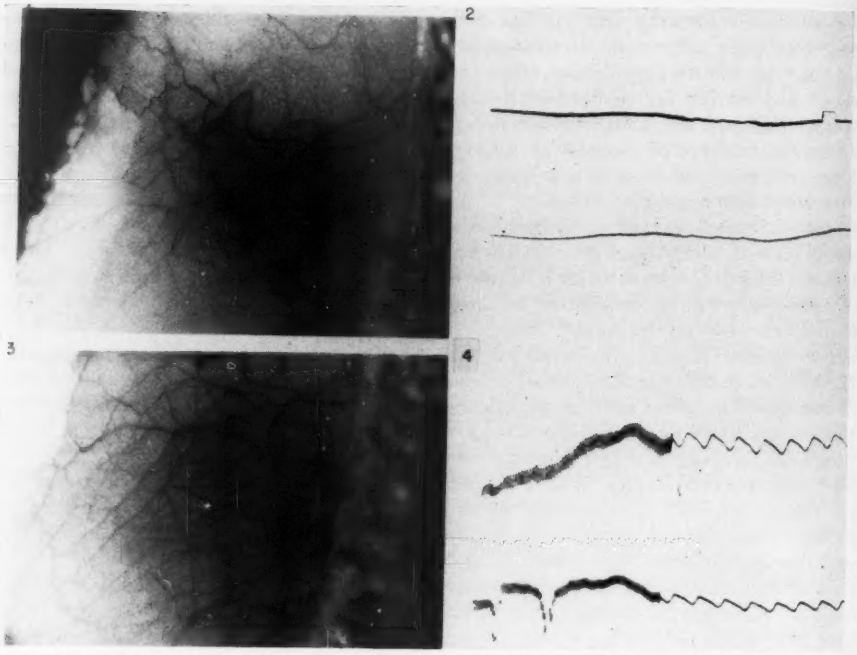

FIG. 1. Photographs of conjunctiva and digital plethysmographs made before and after Ilidar infusion in patient with vasospastic disease. (*1*) Conjunctiva before infusion; (*2*) plethysmograph before infusion; (*3*) conjunctiva after infusion; (*4*) plethysmograph after infusion.

with an LF oscillograph was used for all digital plethysmography). The blood pressure was recorded, both with the standard sphygomanometer and with a direct-writing aneroid type oscillograph (Gesenius and Keller Model), which was also used to record the pulse amplitude at varying levels on the extremities. Graphic recordings of the skin and muscle temperatures of the extremity at multiple sites were obtained by means of a multi-lead thermocouple, electronically connected to a Speed-O-Max recording device. The pulse rate was recorded on an electrocardiogram obtained with a standard Sanborn EKG unit. Notation was made of any subjective symptoms or complaints which the patient noted during the control period. The drug infusion was then started, usually in an antecubital vein, and this was continued while the recordings outlined above were carried out. The dose of drug was according to the recommendations of the individual manufacturers. The infusion was allowed to drip at a rate designed to reach its conclusion at the end of a 30-min. period. At the end of the infusion, all of the observations and records which had been obtained during the control period were then repeated. An interval of at least 24 hr. separated each study.

Total volumetric blood studies, using the Evans dye method before and after infusion of the vasodilator drugs, was started late in the investigation and the results are incomplete.

RESULTS

The 15 patients observed in this study represent most of the general classifications of common peripheral vascular diseases. As vasospasm varies considerably according to the different diseases, the group was sub-divided into patients with pure vasospasm, patients with arteriosclerosis, patients with Buerger's disease, and a miscellaneous group which included a patient with essential hypertension who had been subjected to bilateral thoracolumbar sympathectomies, and another who had post-traumatic causalgia.

Vasospastic disease. In the group of 6 patients who had vasospastic disease, there was no single drug which produced uniform results. The responses not only varied from one patient to

another with identical symptoms, but also varied from one extremity or phalanx to another. The digital plethysmographs (fig. 2) showed greatest response to Ilidar, next Priscoline, followed by little response to Arlidin and Drug 5029. Priscoline was not administered to one patient known to have a duodenal ulcer.

The muscle temperature (fig. 3) was more responsive as a whole to Arlidin and Priscoline, but this was inconsistent and actually varied between limbs as well as among patients. In one case, Ilidar caused increase in muscle temperature from 2 to 25°F. as contrasted to a decrease of 0.5–2° following the administration of Arlidin and Drug 5029.

The skin temperatures (fig. 3) were decreased in more than one half of the group of patients after infusions of Arlidin and Drug 5029. These temperatures were increased in 4 of 6 patients after Ilidar and Priscoline. The skin temperatures were decreased by all drugs in one patient who, at the same time, showed a good muscle temperature response to Arlidin and Drug 5029.

The conjunctival vessel responses consistently correlated with the other methods of measuring flow in all drug studies (fig. 1). Where there were poor peripheral changes, there were no appreciable conjunctival changes.

The oscillographs were not affected to any extent by any of the drugs used.

There were few major alterations in blood pressure. In at least one patient during the infu-

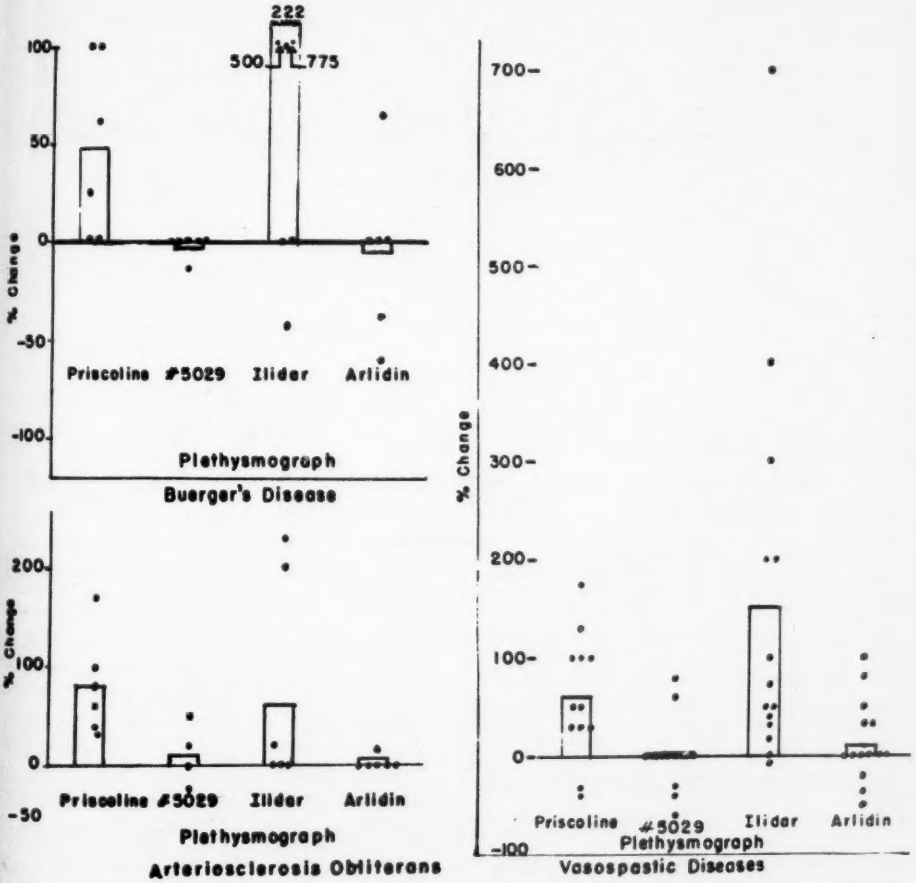

FIG. 2. Digital plethysmographs made after infusions of all drugs in the various types of arterial diseases. The dots represent the actual change and the blocks represent average changes.

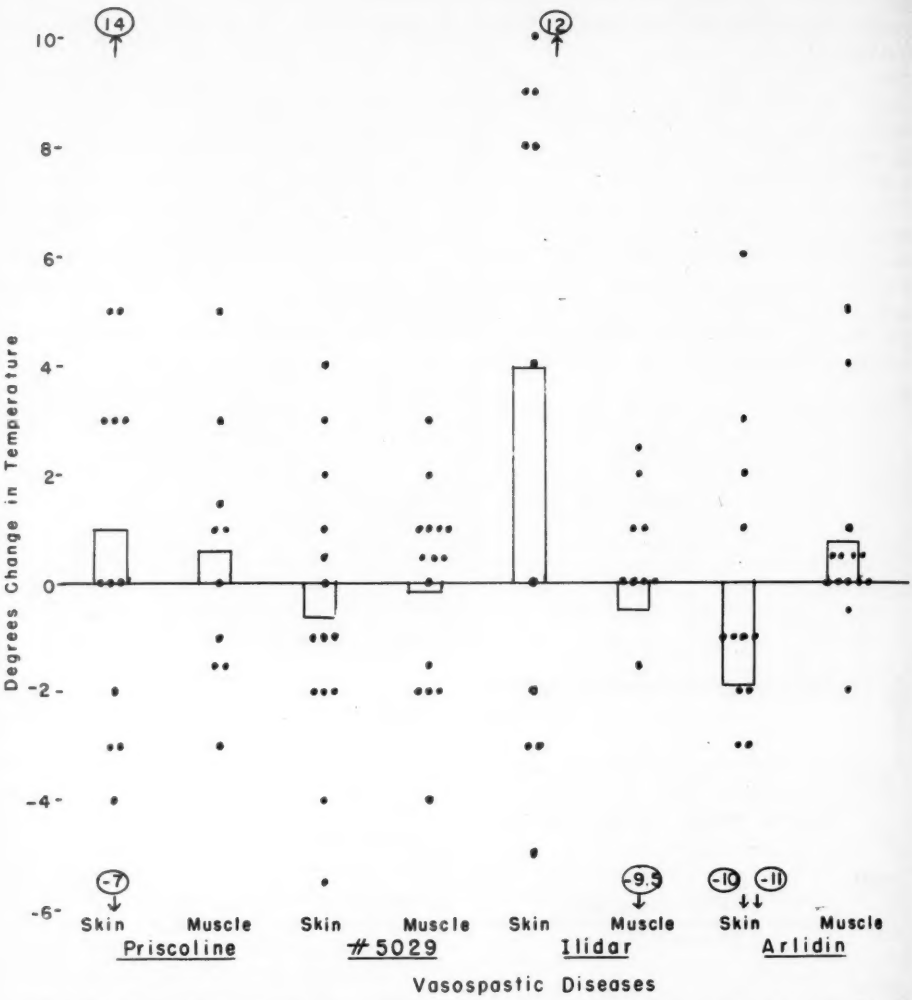

FIG. 3. Muscle and skin temperature changes expressed in degrees F. in the vasospastic diseases. The dots represent actual recordings of temperature while the blocks represent average changes.

sion of all drugs, there were increased blood pressure readings. Generally, however, the blood pressures were diminished. The decreases were averaged as follows: Arlidin, 3 mm.; Ilidar, 9 mm.; and Drug 5029, 5 mm. Hg. In Priscoline studies, there was an over-all increase of 2 mm. Hg.

There were no electrocardiographic changes in vasospastic patients after Arlidin and Priscoline injection. However, there were S-T segment depressions in two patients in whom Ilidar was used, and S-T segment depression and inversion of QRS complex in one patient after Drug 5029.

From an over-all standpoint, Ilidar and Priscoline were the most effective drugs in vasospastic diseases, according to the observations recorded.

Arteriosclerosis. In this group of three patients, there were minimal responses in all studies. Priscoline had more desirable effects on the plethysmograph (fig. 2) and skin temperatures, and showed equally good muscle temperature (fig. 4) response as any of the three other drugs.

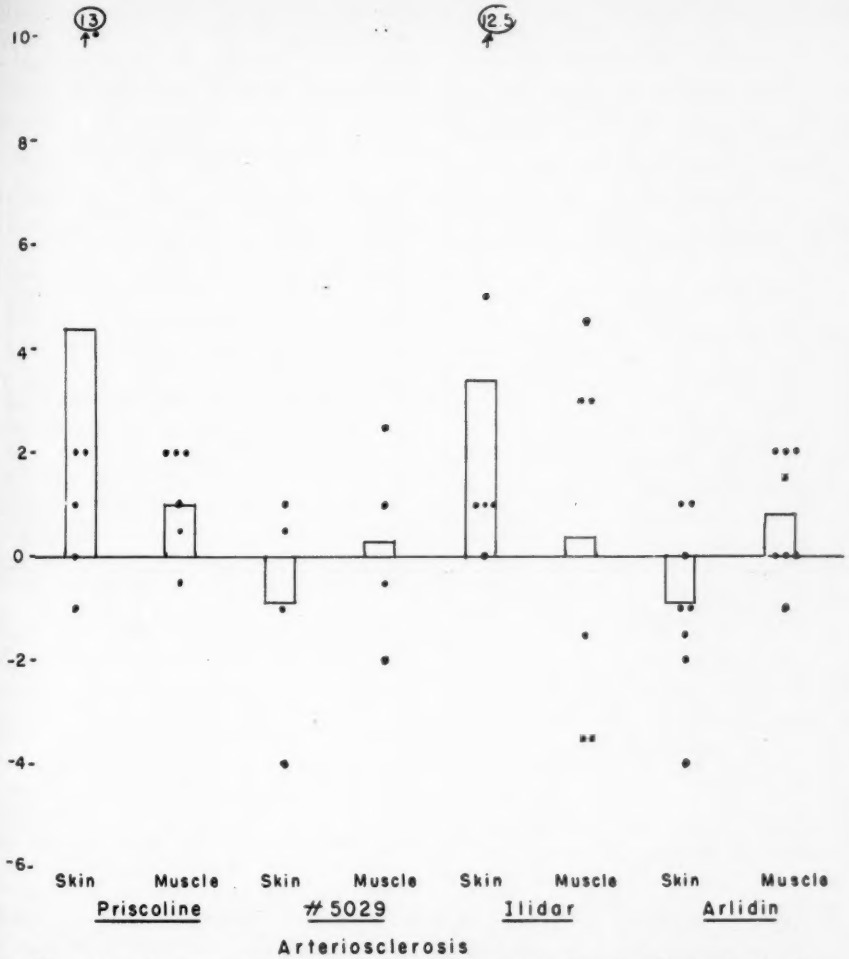

FIG. 4. Muscle and skin temperature changes expressed in degrees F. in the arteriosclerotic diseases. The dots represent actual recordings of temperature while the blocks represent average changes.

The average decreases of blood pressure in mm. Hg. are as follows: Arlidin, 3 mm.; Ilidar, 43 mm.; Priscoline, 2 mm.; and Drug 5029, 3 mm. The decrease in blood pressure in the Ilidar study was caused by the fact that one of the patients had a blood pressure drop from 170/90 to 80/46 mm. Hg. This hypotension was associated with nausea, vomiting and prevented continuation of the study.

Buerger's disease. The plethysmograph responses (fig. 2) were excellent with both Ilidar and Priscoline and negligible in Arlidin and Drug 5029.

In only one patient was there demonstrable increases in muscle temperature (fig. 5). He responded well to Arlidin, Priscoline, and Drug 5029 but reacted unfavorably to Ilidar. Conversely, the skin temperatures increased much more after Ilidar infusion than that of any of the other three drugs.

Here, as with the other groups, the conjunctival studies compared favorably to the other responses.

On the electrocardiograph, one of the patients with Buerger's disease (L. F.) showed S-T segment depression after Arlidin and severe tem-

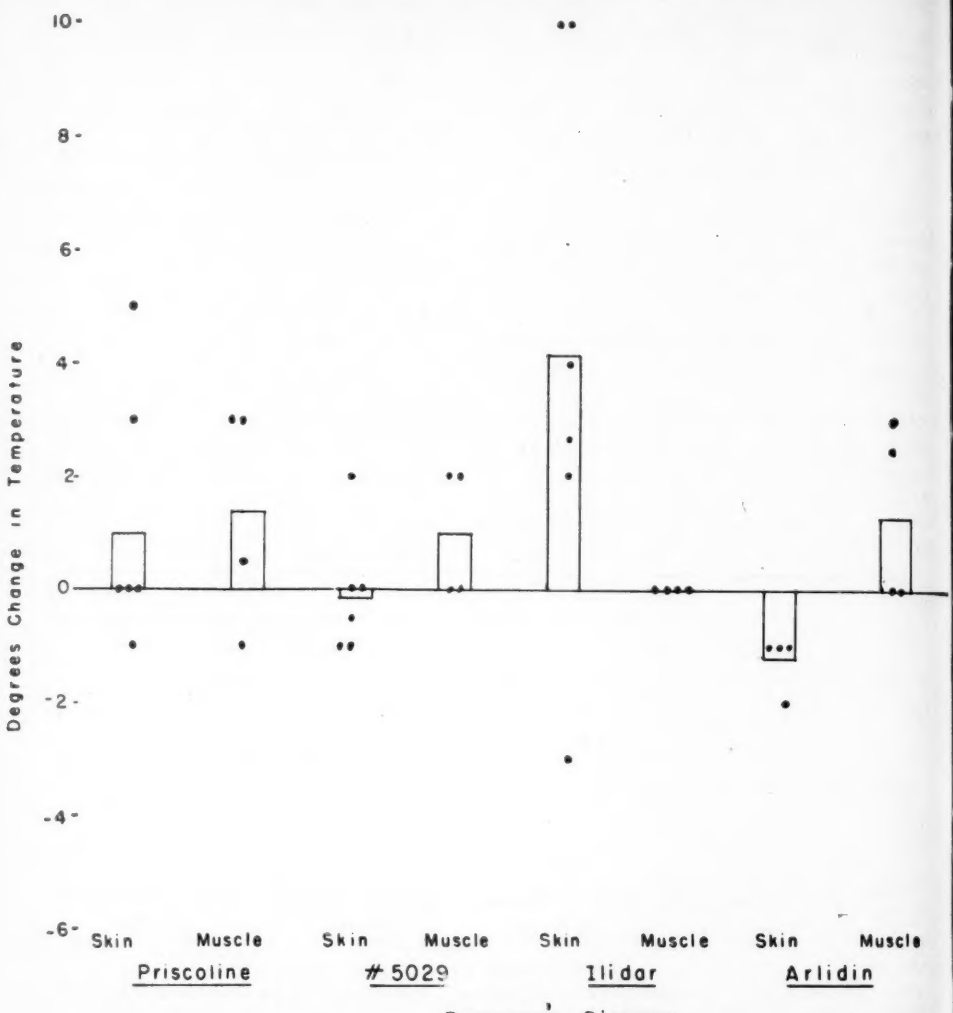

FIG. 5. Muscle and skin temperature changes expressed in degrees F. in Buerger's disease. The dots represent actual recordings of temperature while the blocks represent average changes.

porary heart block with clinical shock after Drug 5029. He demonstrated no abnormal electrocardiograph changes after Ilidar and Priscoline.

*Miscellaneous group.** One of the patients was a 35-year-old female with essential hypertension who had responded well to bilateral thoracolumbar (T-4 through L-2) sympathectomies. She reacted to Ilidar and Priscoline by increase in plethysmographic amplitude, decrease in muscle temperatures, increase in skin temperature and increase in blood pressure recordings. In response to infusion of Arlidin and Drug 5029, there were no changes in plethysmographic tracings, decrease in muscle and skin temperatures, and depression of blood pressure.

The other patient in this group had posttraumatic causalgia of the leg. He showed mini-

* On the charts, these two patients are classified as "vasospastic."

mal evidences of increased peripheral blood flow after Priscoline and Ilidar infusions and no appreciable response to the other drugs. There were no subjective improvements to any of the drugs used.

DISCUSSION

In that all of these patients had identical studies at metabolic rest in a constant-temperature-humidity room, the results following standardized infusion should be easily categorized. This was not found to be true. Patients with identical clinical manifestations responded differently at times and the responses varied from one limb to another. For example, a young female with classical Raynaud's phenenomena of the fingers showed an average decrease in skin temperature of 10°F. in the left fingers and 11°F. in the right fingers after Arlidin, a decrease of 5°F. in right fingers and an increase of 2°F. in left fingers after Priscoline, a decrease by 5° and 2°F. left and right fingers respectively after Drug 5029 and an increase of 5°F. bilaterally after Ilidar.

In general, the greatest over-all response in all studies on all patients were best after Ilidar and Priscoline. This does not mean that either or both were always effective. Arlidin and Drug 5029, as would be expected from their chemical derivation, caused similar responses in the patients, but Arlidin produced less side reaction in the dosage administered.

The average values only compare efficacy of the drugs, and do not indicate absolute value of the drug, *i.e.*, in many of these cases (especially arteriosclerosis and Buerger's disease) the major arteries were mechanically occluded in one extremity and could not possibly respond to a vasodilator, thus lowering the average response markedly. However, associated spasm in the contralateral limb may have been improved by the drug.

The skin temperature and plethysmograph generally correlate better than muscle temperature and plethysmograph suggesting that our plethysmographic method may measure skin flow to digits predominantly, and muscle flow may respond differently.

In several cases, drug reaction (severe nausea) was so marked as so obviate post-infusion recordings. Hence the discrepancy of the number of tests among the drugs.

It is known that some specific vasodilators react differently on separate groups of arteries such as the cerebral, renal, and coronary systems. From this study, the vessels of the extremities did not react uniformly, in general, and even reacted differently in one extremity from another in the same person. This suggests a "chemoreceptor mechanism" in each major system of arteries, that not only varies in response in different persons but varies from time to time in the same person.

SUMMARY

1. Fifteen patients with representative peripheral vascular diseases had detailed laboratory studies made before, during, and after infusions of four vasodilators.

2. None of the drugs showed consistently good results in all types of vascular diseases.

3. The responses not only varied between persons with similar diseases but varied from one extremity to another in the same person.

4. Ilidar and Priscoline proved most effective in the majority of patients, especially from the detailed plethysmographic and temperature studies.

5. There is excellent correlation between the effects of these drugs on the conjunctival vessels and the small vessels in the extremities, thus affording an easily accessible source of photographable vessels to be used in the testing of vasodilator groups.

6. Vasodilators which increase cardiac output must be administered cautiously in patients with heart disease.

7. Additional studies in the animal laboratory with special regard to the effects of these drugs on the cardiac contractile force are indicated.

8. It is postulated that each major system of arteries has its own "chemoreceptor mechanism" causing tremendous variations in the effects of vaso-reactive drugs in different parts of the body at the same time.

Acknowledgement. The authors would like to express their appreciation for the technical aid of Miss Ann Boone, R.N. and Mrs. Sue Walters, R.N. for their assistance in this study.

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GASTROJEJUNAL ULCER AND GASTROJEJUNOCOLIC FISTULA*

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The development of a gastrojejunal ulcer, and in an advanced case, its extension into the colon to form a gastrojejunocolic fistula, represents a failure of previous surgical treatment of peptic ulcer or it means that the patient has developed, after initial treatment, a more severe ulcer diathesis for which the initial treatment is inadequate.

Recurrent ulcer develops in about 15 to 20 per cent of cases when gastroenterostomy alone has been the surgical treatment for duodenal ulcer. It develops in about 3 to 5 per cent of cases after a 60 to 70 per cent subtotal gastric resection. Thus its occurrence has a significant rate, and the high rate of its development after gastroenterostomy alone has led to the discard of that procedure as a primary surgical treatment for the complicated duodenal or gastric ulcer. It is highly probable that the incidence of gastrojejunal ulcer is higher than figures quoted in the literature because diagnosis of gastrojejunal ulcer may be difficult, and there are many patients who have episodes of gastric hemorrhage or abdominal pain who never have a definitive proved diagnosis

At the Sanders Clinic in the years from 1936 through 1957, more than 1000 cases of duodenal ulcer have been surgically treated. During this same period of time, 51 cases of gastrojejunal ulcer have been surgically treated and 5 of these cases had a gastrojejunocolic fistula. This represents an incidence of 5 per cent of the total cases of duodenal ulcer which have been treated by operation.

We agree with Priestley and Gibson[1] that a proved gastrojejunal ulcer is nearly always a surgical problem, and unless some medical contraindication or other complicating factor is present, we usually advise surgical treatment. Thus these 51 cases represent the majority of our experience with proved gastrojejunal ulcer.

Forty-one of these patients were men and ten were women. Their ages ranged from 22 to 72 years with the greatest incidence in the fourth, fifth, and sixth decades of life.

Thirty-six cases had had gastroenterostomy as prior treatment for duodenal ulcer. Nine had had gastric resection; five had had vagotomy and gastric resection, and one had had a pyloric exclusion operation.

The time interval was variable between the above mentioned surgical procedures and the development of further trouble. In the gastroenterostomy cases, nine developed trouble within one year or less, while fourteen did not develop recurrent ulcer symptoms until more than ten years had elapsed.

Thirty-two patients were treated by take-down of a previous anastomosis and gastric resection. Ten patients were treated by vagotomy. One of these ten also had an enteroenterostomy, and in one the gastroenterostomy was taken down and local excision of the ulcer was carried out. Three patients had resection of a gastroenterostomy with local excision of the stomal ulcer, and in one of these a new gastroenterostomy was done Three had closure of free perforation of a gastrojejunal ulcer; two had vagotomy and gastric resection combined, and one had vagotomy and repeat resection of the stomach (table 1).

Seven of the aforementioned patients had recurrence of their gastrojejunal ulcer and had further surgery. Four of these had gastric resection after previously having had vagotomy and gastroenterostomy. One had vagotomy after having previously had gastric resection. One patient had gastrectomy after a previous local resection of a gastrojejunal ulcer and one case had repeat resection (table 2).

SYMPTOMS

The symptoms of gastrojejunal ulcer are those of pain, hemorrhage, or are obstructive in nature. The pain is usually located in the epigastric or mid-abdominal area and occasionally may be located to the left of the midline. It is usually similar to that of peptic ulcer, but in contrast to the pain of duodenal ulcer frequently there is no

* Sanders Clinic, Memphis, Tennessee.

Presented during the Baltimore Assembly of The Southeastern Surgical Congress, March 10–13, 1958, Baltimore, Maryland.

TABLE 1

Treatment of gastrojejunal ulcer

	No. of Patients
Resection of ulcer, gastroenterostomy, and two-thirds of stomach*	32
Vagotomy	10
Closure of free perforation	3
Vagotomy and gastric resection	3
Local excision of gastrojejunal ulcer	3
Total	51

* Includes 5 patients with gastrojejunocolic fistula.

TABLE 2

Treatment of recurrent gastrojejunal ulcer

Gastric resection after previous vagotomy	4
Gastric resection after previous resection	1
Gastric resection after previous local excision	1
Vagotomy after previous gastric resection	1
Total	7

food relief, and in some cases, there is aggravation of pain after ingestion of food.

Gastrointestinal hemorrhage after a previous resection or gastroenterostomy for ulcer usually means a gastrojejunal ulceration except in the period immediately following surgical intervention. Hemorrhage may occur with or without pain. Hemorrhage from gastrojejunal ulceration is usually mild in degree and seldom is it necessary to do an emergency operative procedure because of the danger of shock or exsanguination. Exceptions to this occur as illustrated by one patient who died of hemorrhage and another in whom emergency operation was done because of exsanguinating hemorrhage.

Obstructive symptoms of nausea, vomiting, fullness, excessive gas and belching are often present and frequently in such instances one finds that the ulcer has narrowed the gastroenterostomy by inflammation and edema. One must separate from this group those patients who have dumping syndrome symptoms following previous resections. Eighteen patients in our series had hemorrhage as their predominant complaint. Twenty-seven patients had abdominal pain as their predominant complaint, twenty-two had nausea, indigestion, and vomiting as predominant complaints. Three patients had sudden severe abdominal pain which signalled the presence of an acute free perforation of a gastrojejunal ulcer. These cases are discussed in subsequent paragraphs.

The five patients who had associated colic fistulas had diarrhea and weight loss associated with their symptoms of abdominal pain and tenderness.

Physical examinations were not significant unless some unusual features were present. For example, one patient with a large incisional hernia and gastrojejunal ulcer had a large intra-abdominal mass which could be palpated through the hernia. This was thought to be a carcinoma, but proved to be a large ulcer with inflammation in surrounding tissues.

Abdominal tenderness was present in many, and signs of anemia and blood loss were present in those who had severe bleeding.

In those instances when gastrointestinal x-rays were made, the findings were positive for gastrojejunal ulcer in 20 patients, suggestive of ulcer in 16 patients, and negative in 8 patients.

Gastric acidity, in 23 patients, varied from 20 degrees to 160 degrees. Unfortunately the number of patients in which this measurement was made was too few for reliable conclusions in our study. It may be said, however, that the level of acids alone is insufficient evidence upon which to base a conclusion as to the presence or absence of gastrojejunal ulcer.

COMPLICATIONS OF GASTROJEJUNAL ULCER

Acute free perforation is a rare complication of gastrojejunal ulcer. In the series of Walters, Chance, and Berkson,[2] there were only 2 patients in a series of 301 cases studied who had been seen with acute free perforations of gastrojejunal ulcers. However, 8 of their patients had histories of previous free perforations. In our series, 3 patients had acute free perforation into the peritoneal cavity. In all of these the previous surgical procedure had been gastric resection. In 2 of these, the anastomosis was antecolic, and in the other the anastomosis was retrocolic. The patient who had the retrocolic anastomosis also had a perforation into the colon with a gastro-

jejunocolic fistula present. The 2 patients who each had an antecolic anastomosis were treated by surgical closure of their perforations with recovery. The third patient developed an intra-abdominal abscess which was later drained. In this case, a second free perforation occurred 1 month later and was treated by total gastrectomy. This patient died, however, of peritonitis.

In 5 cases the gastrojejunal ulcer had perforated into the colon producing a gastrojejunocolic fistula. The case associated with a free perforation has been described. The other 4 patients had symptoms of diarrhea, weight loss, abdominal pain, and malodorous eructations. Each of these had previously had a posterior gastroenterostomy. They were diagnosed by barium enema roentgenogram and all were treated by resection of the gastroenterostomy, gastric resection, and closure of the colic fistula as a one stage procedure. One of these patients died of pneumonia and atelectasis; this death was in 1936 before the use of chemotherapy. The other 3 patients have been operated on in recent years and have all done well with uneventful recoveries and good results.

The incidence of the complication of hemorrhage has been discussed in preceding paragraphs.

RESULTS

A total of 58 surgical operations were done for gastrojejunal ulcer or its complications. Two patients died postoperatively, which is a mortality rate of 3.4 per cent for our group of cases. One of these died of atelectasis and pneumonia following a one-stage resection and repair of gastrojejunocolic fistula. The other death was caused by peritonitis following total gastrectomy. This patient had previously had free perforation both into the peritoneal cavity and into the colon.

Including the 2 postoperative deaths, 9 of the 51 patients are dead. One of these died of hemorrhage when a recurrent gastrojejunal ulcer eroded into the splenic artery. Two died of anemia, malnutrition, and narcotic addiction. The remaining 4 died of causes unrelated to their prior surgical operations or to their ulcers.

Follow-up studies for a period of from 1 to 20 years were obtained from 44 patients. This was by questionnaire or by examination and includes the patients who are known to be dead. The results of treatment will be considered according to the procedures which were done.

The results were classified as excellent, satisfactory, and unsatisfactory. They were considered in the excellent group if the patient was essentially asymptomatic, if he could eat a reasonably sized meal, and if he had no evidence of malnutrition or anemia. The satisfactory group included those patients who were satisfied with the result, but in whom either anemia existed or in whom there were some complaints referable to the gastrointestinal tract. The unsatisfactory group included those in whom definite recurrences occurred in or whom pain or hemorrhages suggested recurrence, and also those with severe dumping syndrome symptoms, or any who stated they were dissatisfied with their results. Also, those deaths which were considered to have been caused by complications secondary to treatment of the ulcer were included with the unsatisfactory classification.

The results will be considered in relation to the procedures which were done for treatment. The majority of cases were treated either by gastric resection with take-down of the gastroenterostomy, by vagotomy and gastroenterostomy combined, or by vagotomy plus gastric resection (table 3).

As indicated in table 3, we had 26 patients in whom gastric resection with resection of the stomach ulcer and performance of a new gastrojejunostomy were done. Seventeen of these obtained excellent results; 5 were satisfactory, and 4 had poor results. In 12 patients vagotomy was added to gastroenterostomy as a method of treatment of gastrojejunal ulcer with 2 excellent results and 10 poor results (table 3).

Nine patients had vagotomy and resection as their final definitive treatment; this being done as subsequent procedures in some cases. Of this group, 4 had excellent results, 1 was satisfactory,

TABLE 3

Results of treatment of gastrojejunal ulcer

Treatment	No. of Procedures	Results		
		Excellent	Satisfactory	Unsatisfactory
Vagotomy and gastroenterostomy	12	2		10
Gastric resection	26	17	5	4
Vagotomy and resection	9	4	1	4
Miscellaneous	6	1	3	2

and 4 were unsatisfactory (table 3). All of these patients had had at least a 65 per cent gastric resection in association with vagotomy. The primary cause for an unsatisfactory result in both the gastric resection cases and in the vagotomy plus gastric resection cases was anemia and malnutrition plus symptoms of the dumping syndrome. In only 1 case was the recurrence of the ulcer the cause for the unsatisfactory result.

DISCUSSION

In this series of cases of gastrojejunal ulcer, there were 12 cases that had gastroenterostomy and vagotomy. These were done either as primary treatment for duodenal ulcer or for stomal ulcer appearing after primary gastroenterostomy. In only 3 of these were vagotomy and gastroenterostomy the final definitive procedures. Two of them obtained an excellent result and 1 had an unsatisfactory result. These were cases that initially had a gastroenterostomy followed by development of a gastrojejunal ulcer and then later they had vagotomy as the method of treatment. In 9 cases a gastrojejunal ulcer either developed in spite of vagotomy and gastroenterostomy or a gastrojejunal ulcer was not cured or recurred. In these a gastrectomy was later performed or the patient continued to have trouble (table 3).

There is a high rate of development of gastrojejunal ulcer in patients who have had gastroenterostomy for duodenal ulcer. This may develop many years after the initial surgery. In our series, 14 patients developed trouble after 10-year symptom-free intervals, and 1 patient did not have trouble develop for 27 years. The risk of development of gastrojejunal ulceration after gastroenterostomy is about four times greater than after gastrectomy. The frequency of recurrent ulcer after vagotomy and gastroenterostomy is difficult to assess, as this operation had been done only in recent years. Until large numbers of patients have been followed for at least 10 to 15 years, one will not be able to completely evaluate this procedure.

The fact that in this group of 51 cases there were 12 who had vagotomy and gastroenterostomy and later developed either a gastrojejunal ulcer or the recurrence of one leaves us with the opinion that this combination is a poor one for treatment of duodenal ulcer or gastrojejunal ulcer. Vagotomy in association with gastric resection may prove to be a satisfactory operative treatment.

Gastrojejunal ulcer and gastrojejunocolic fistula should be suspected in anyone with a history of previous surgical operation for peptic ulcer in whom abdominal pain, hemorrhage, nausea or vomiting, weight loss, or diarrhea occurs, either as a single symptom or in combination. Physical findings are usually minimal, but roentgen findings are positive or suggestive in 80 per cent of cases. Gastric acids may be elevated but neither achlorhydria nor absence of roentgen findings are sufficient to rule out gastrojejunal ulcer.

The complications of gastrojejunal ulcer are hemorrhage, obstruction, free perforation, and perforation into the colon. Hemorrhage is the most frequent complication and usually means gastrojejunal ulcer is present if there is an antecedent history of gastroenterostomy or gastrectomy for ulcer. Free perforation occurred in three cases in this series. Usually this is in associa- with with an antecolic anastomosis, but in one case it was free into the peritoneal cavity and also into the colon. In that case a retrocolic anastomosis had been done.

Five cases had a gastrojejunocolic fistula one of which was associated with a free perforation as mentioned. One of these patients died after operation and one died a few months after an operation for a second free perforation.

We believe that with modern techniques and antibiotics, the treatment of choice for gastrojejunocolic fistula is a one-stage resection of the stomach, the ulcer, and closure of the colonic fistula. Our last three cases done in recent years have all had uneventful postoperative courses.

One of our patients had a proved pancreatic islet cell adenoma in association with a recurrent gastrojejunal ulcer which eroded into the splenic artery and caused death from hemorrhage. This emphasizes, in our minds, the need for searching for such tumors when operating for gastrojejunal ulcers. This case which fits the so-called Ellison-Zollinger syndrome will be reported elsewhere in detail.

Our results indicate that 85 per cent of cases will obtain excellent or satisfactory results after gastric resection or repeat resection for gastrojejunal ulcer following previous gastroenterostomy or gastrectomy.

Our experience with vagotomy plus gastrec-

tomy has yielded excellent to satisfactory results in 55 per cent of cases. Vagotomy and gastroenterostomy yielded about 16 per cent excellent to satisfactory results when done for gastrojejunal ulcer (table 3). In most of these a vagotomy was done after a gastroenterostomy.

SUMMARY

1. In a group of approximately 1000 surgically treated patients with duodenal ulcer, 51 have developed gastrojejunal ulcers. Five of these had gastrojejunocolic fistulas.

2. Thirty-six of the 51 patients developed a gastrojejunal ulcer after gastroenterostomy; 10 of these 36 had had vagotomy added to the gastroenterostomy. Nine cases had had gastric resection as their primary surgery, 5 had vagotomy and gastric resection, and 1 a pyloric exclusion operation.

The most common symptoms in this series were abdominal pain, nausea, vomiting, hemorrhage, weight loss, and diarrhea.

4. Roentgen findings are positive or suggestive in 80 per cent of cases.

5. Treatment, in most cases, was by gastric resection or by gastric repeat resection when a previous resection had been done. This procedure yields best results (85 per cent excellent to satisfactory) as compared with 55 per cent excellent to satisfactory results with vagotomy and resection, and 16 per cent excellent to satisfactory results with vagotomy plus gastroenterostomy.

6. The total postoperative mortality rate was 3.4 per cent or two deaths. Each of these patients had a gastrojejunocolic fistula, and one had a free perforation with peritonitis before operation.

7. One patient had an associated pancreatic islet cell adenoma in association with a recurrent gastr jejunal ulcer. This was discovered at autopsy when the patient died following hemorrhage caused by erosion of the splenic artery from a recurrent gastrojejunal ulcer. It is possible that some of the other patients in this series may have had this syndrome but did not have discovery of the pancreatic tumor. Our policy now is always to explore carefully the pancreas when operating for gastrojejunal ulcer.

8. Three patients had free perforation of the gastrojejunal ulcer into the abdominal cavity, and one of these had free perforation on two occasions. This unusual complication was treated satisfactorily in two cases by simple closure of the perforation. In one case the existence of peritonitis led to an abscess formation, and a second free perforation later was treated by a radical resection; however the patient died of peritonitis in the period following surgery.

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UNUSUAL COMPLICATIONS OF BILIARY TRACT SURGERY —THEIR AVOIDANCE*

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Operations upon the biliary tract are very common because of the frequency with which the gallbladder becomes involved in either stone formation, inflammation, or both. Although cholecystectomy is considered a reasonably simple procedure compared to other operations within the abdominal cavity, the gallbladder and ducts in certain individuals may be difficult to expose. Because of the delicacy of duct structure and the fact that the common duct must be considered to be a vital organ, operative injury may have very serious consequences.

Probably every surgeon of experience has inadvertently injured the common duct at some time during his career. However, the important consideration is that the trained surgeon will recognize the injury at the time, will be able to repair the damage immediately and consequently serious complications will be rare. However, if the injury is not recognized at operation but is discovered only when jaundice appears, repair may be very difficult because of adhesions, inflammation and infection and the poorer condition of the patient generally.

Extensive studies and illustrations of the anatomy of the extrahepatic biliary apparatus have been made. If there is one characteristic feature of these studies, it is variation. Variation does not imply simply the anomalies which occur in this region; it implies also the differences in body build and organ situation as well as the distortion caused by the disease process present. Therefore, it would seem that a knowledge of surgical anatomy implies a dynamic concept of how to expose whatever is encountered, whether it is an anomaly, a relatively inaccessible gallbladder, or extensive disease.

Avoidance of local surgical complications in the area of the biliary tract, then, depends almost completely upon exposure. This statement is of course true for all surgical operations. Exposure begins with the incision, and the incision for exposure of the gallbladder and biliary ducts must not be "routine." It should depend upon the size, weight and body configuration of the patient, as well as upon the problem at hand. Whereas a subcostal incision severing the right rectus muscle may be entirely adequate for a simple cholecystectomy, it may not be adequate at all for a difficult common duct exploration. Control and dissection of the common duct region is perhaps most effectively performed when the surgeon—on the patient's left—inserts the fingers of his left hand beneath the hepatoduodenal ligament with the thumb on top. In this position there must be room for the left wrist and this is best accomplished by making a long paramedian incision. For complicated secondary operations on the biliary tract where there has been repeated operative trauma, it may be almost essential to make a thoracoabdominal approach with upward displacement of the liver and a more lateral attack on the hepatoduodenal ligament. Probably the common denominator of all good incisions is their length. It is noteworthy that when an attending surgeon scrubs in after a second year assistant resident has opened the abdomen, the first thing the surgeon usually does is to extend the incision two to three inches. It is also noteworthy that the majority of cases which are referred following injury to some structure show a pitifully short, original incision. Stewart and Mallo[1] have written an interesting article on the importance of anatomical variation in abdominal surgery. Granting the preceding remarks it must be emphasized in addition that good assistance and skillful anesthesia are basic necessities for good exposure.

To illustrate these points certain selected, unusual, complications of biliary tract surgery have been chosen. From the ideal standpoint all of these complications were preventable. Three of these cases were referred from other hospitals

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Presented during the Baltimore Assembly of The Southeastern Surgical Congress, March 10–13, 1958, Baltimore, Maryland.

following the complication; the last two were the author's own cases.

CASE REPORTS

Case 1. The first patient is that of a 48-year-old man who was operated upon electively for gallstones. During the course of the cholecystectomy serious bleeding was encountered, the source of which, even with added help and exposure, could not be found. The wound was packed with gelfoam and gauze and the operation terminated. The packing was progressively removed each few days. On the 20th day the patient suddenly bled copiously, accompanied by serious shock. When first seen by me the shock had been controlled by transfusions and he was ready for operation. Since there had been considerable bile and purulent drainage throughout his postoperative period, exposure was quite difficult. There was no active bleeding, so no simple clue was present to aid in definitive control. After a thorough unsuccessful search for a possible bleeding point it was felt that surgery should be terminated and packing was again inserted and new drains left in for bile drainage. Four days after this the patient again went into shock from external hemorrhage and this time it was very apparent that the bleeding point had to be found. It was finally located in the right hepatic artery (fig. 1) and the reason for the recurrence of bleeding was apparent. The injury to the vessel was not complete; this situation prevented the ends of the vessel from retracting, thus allowing a firm clot to form. The artery was severed and ligated. The patient recovered, but a bile fistula persisted. This was repaired after a few months by the late Howard Gray, and the patient is well.

Comment: This illustrates the importance of accurately identifying the position and amount of injury at the time of the operation itself. The author himself fell into the same pitfall at the first operation following hemorrhage. The presence of a partially injured artery still on the stretch should have been suspected because of the frequency with which this injury was seen during the war, resulting in recurrent hemorrhage

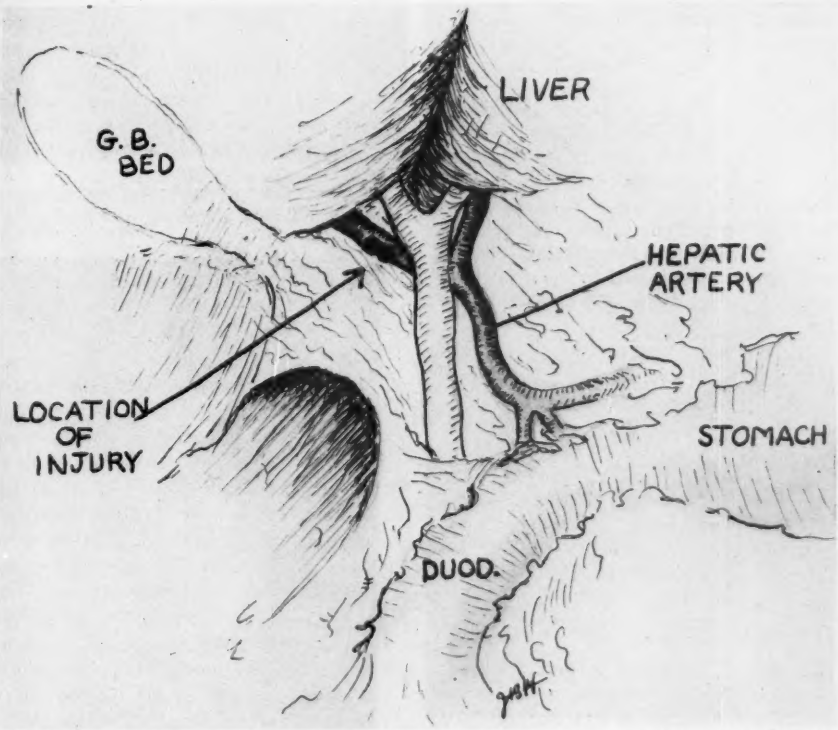

FIG. 1. (Case 1.) Diagrammatic sketch showing approximate location of injury to right hepatic artery. The artery remains on the "stretch."

or the formation of pulsating hematomas. Whereas hemorrhage from a severed cystic artery would probably be controlled permanently by packing, in this instance, there was little chance of a permanent result by this method of control.

Case 2. The second case is that of a woman of 40 years of age who had an elective cholecystectomy for solitary stone. She said that she felt badly throughout her postoperative period, but was discharged from the hospital on her eighth day. She was readmitted on the ninth day because of pain and fullness in the upper abdomen and mild jaundice. She was treated expectantly and was told that the bile had to be thinned out. She became discouraged with increasingly severe symptoms for two and one-half weeks and was subsequently referred to the author. On admission she was quite ill, with a markedly distended and tense upper abdomen, abdominal pain, some difficulty in breathing, and mild jaundice. Intravenous cholangiography was not successful but the plain x-ray film (fig. 2) suggested a globular mass filling the entire upper abdomen. This woman was explored and immediately upon incising the peritoneum greenish-brown fluid escaped under great pressure and 12,000 cc. were evacuated. The incision was then enlarged and the cavity explored. The diaphragm and liver were superior, the stomach posterior, identified only by feel since a greenish, shaggy membrane covered all structures. The mass was limited below by the transverse colon which was adherent to the abdominal wall and exploration in the region of the common duct could not be carried out without violating the intactness of the cavity. This was felt unwise, and th operation was terminated by marsupializinge the sac and inserting drains. The patient obtained immediate relief, and except for a few days during which she had some nausea and vomiting, her postoperative course was quite smooth. Jaundice disappeared entirely in about two weeks and the patient went home with minimal drainage from the drain site. The stools were brown. She has remained well for the past four months.

Comment: It was feared that this represented injury to the common duct with a leak into the upper abdomen, but the patient's course revealed

FIG. 2. (Case 2.) Plain upright film of abdomen showing large rounded mass in upper abdomen.

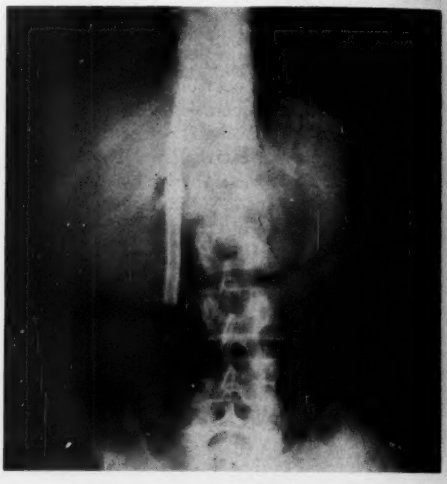

FIG. 3. (Case 3.) Plain film of abdomen showing position of T-tube placed 12 years previously.

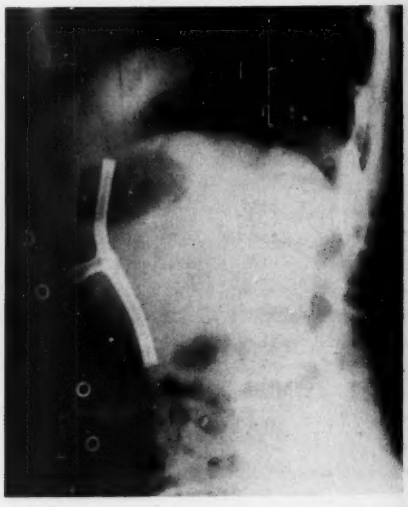

FIG. 4. (Case 3.) Same as figure 3; lateral view.

that probably a tie slipped off the cystic duct. The complete walling off was very fortunate. The patient was quite positive that no drain had been used postoperatively and it has since been established that this was the case. A drain for a few days postoperatively would probably have prevented the complication.

FIG. 5. (Case 4.) Preoperative cholecystogram showing a second opacity in addition to the usual gall bladder shadow.

FIG. 6. (Case 4.) Postoperative cholangiogram showing intrahepatic sac.

Case 3. The third case is that of a woman of 65 who was operated upon for gallstones 12 years previously. There followed two months of jaundice and itching and a second operation was performed. Eight months later a third operation with insertion of a T-tube was necessary. At this time the patient was told that the tube should never come out during the rest of her life. Several years went by; the surgeon who had operated upon her died; and the doctor in charge of her case cut the tube off at skin level hoping that it would pass, or that the fistula would heal. The patient went along for the next ten years with a draining sinus, and with occasional bouts of jaundice, fever and chills. During one of these attacks she was seen by us and a plain x-ray film showed the tube (figs. 3 and 4). This large tube, then, had been in the patient's abdomen for 12 years. Exploration was carried out in December, 1952. Interestingly enough the sinus tract from the skin led down to the region of the pylorus. Upon opening the stomach the tube could be seen protruding into the antral portion and up

FIG. 7. (Case 4.) Postoperative cholangiogram showing condition of common duct and anomaly after operation 4½ years later. (Note pancreatic duct.)

toward the liver. After this removal an exploratory finger could be placed in the sinus, but no bile was obtained and probing revealed no duct. Since the position of the tube was not in the expected area of the common duct, since the patient had not been severely jaundiced, and since exploration of the hepatoduodenal ligament would have been very hazardous, the operation was terminated after closure of the stomach. This patient has remained well for the past five years, with occasional temporary attacks of mild jaundice and fever, controlled by antibiotics.

Comment: This is a remarkable example of the body's ability to tolerate a foreign body for a long period. The tube when removed was not in the common duct and it is possible it never had been. In any event its removal resulted in restitution of health.

Case 4. The fourth case represents an anomaly. The patient was a woman of 64 with typical attacks of gallbladder colic and common duct obstruction with jaundice. The preoperative cholecystogram (fig. 5) showed what apparently was a gallbladder folded back upon itself; or a true double gallbladder. At operation the gallbladder was found to be of normal shape and free of stones. The common duct was thickened and obviously full of stones. The stones were completely cleaned out and the gallbladder removed. A postoperative cholangiogram revealed the true situation (fig. 6) and it was only then apparent that there was an ectasia or diverticulum of the right hepatic duct,

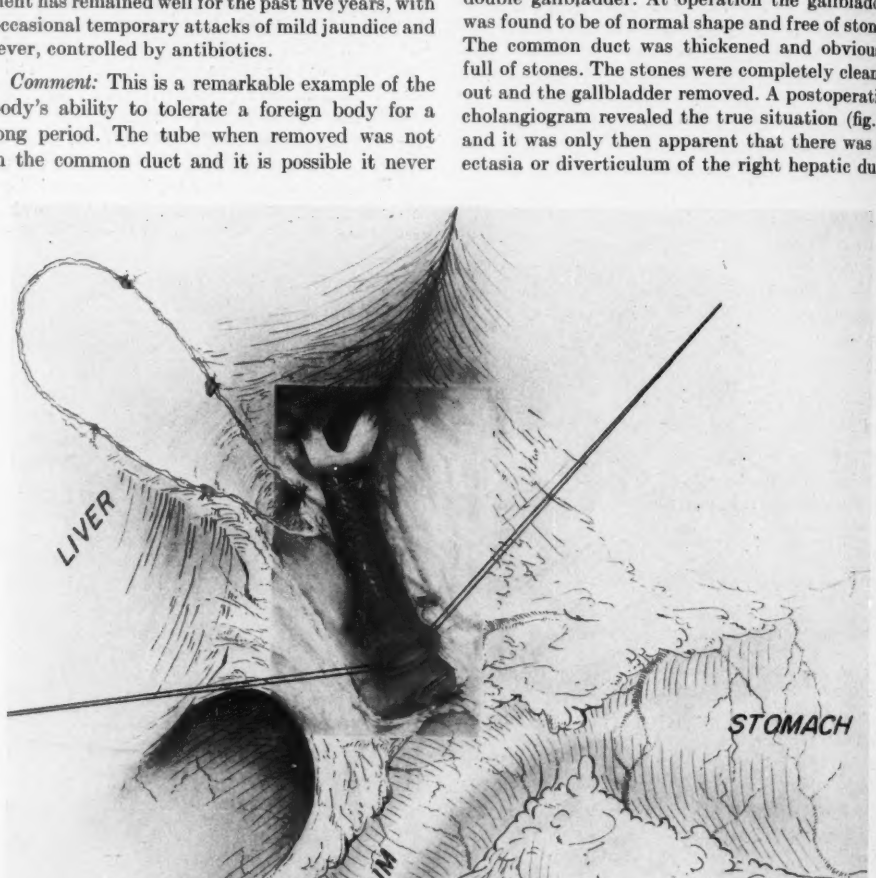

FIG. 8. Drawing of situation in case 5. Gangrene of the common duct 30 min. after simple exposure and preparation of 3 cm. of duct. (Duct was not transected when gangrene was noted.)

or an anomalous second gallbladder. This pouch inside the liver had, of course, been the source of the stones in the common duct. Considerable apprehension was entertained as to whether stones had been left in this area since it was not explored at the time of operation. The patient remained well for six months but then had a severe attack of pain and jaundice. The situation being known she was reoperated upon. The upper sac was reached with probes and scoops and numerous stones removed from it as well as a great deal of mud; and many stones were removed from the common duct itself. An interesting immediate complication occurred in this woman; as the resident was closing he suddenly noticed that blood was dripping rather briskly from the T-tube. The surgeon re-scrubbed and re-opened the abdomen and found the bleeding to arise from a small artery high up in the wall of the inside of the duct. This was easily controlled. Four and one-half years later a third operation became necessary for choledocholithiasis, the origin of the stones again being from the intrahepatic anomaly. Figure 7 shows the large size of the common duct at this time. The patient has been well for three years.

Comment: This represents a congenital anomaly and an error on our part for not recognizing at the first operation the inconsistency between a totally empty and relatively uninflamed gallbladder and a common duct full of stones. We believed the cholecystogram was an artifact. Obviously in this case an operative cholangiogram would have solved the problem and probably would have obviated one of the two subsequent operations.

Case 5. The final case is that of a female physician, in her mid forties, who was operated upon for a suspected malignancy of the distal end of the stomach or pancreas. Carcinoma of the pancreas was found, proved by frozen section, but it was clearly non-resectable. Since the common duct was slightly dilated and just beginning to be obstructed, a side-tracking of the common duct and a gastrojejunostomy were decided upon. The common duct was exposed for a distance of about 3 cm., was not transected, and was covered over with a gauze pack. An isolated Roux-Y limb was then constructed from the jejunum, requiring about 30 minutes. The limb was brought up behind the colon and the pack removed from the area of the common duct. Astoundingly enough it was immediately apparent (fig. 8) that during this short period of time the common duct had become necrotic and was undergoing autolysis. The illustration indicates that the two hepatic ducts were easily visualized in the hilum of the liver, but actually this was not the case and any attempts at dissection in this area seemed very dangerous because of the friability of the necrotic tissue. So the duct was severed at its distal portion and then guided in to an incision in the jejunum on the end of a suture (fig. 9). Nothing seemed feasible except to suture the seromuscular coat of the jejunum to the capsule of the liver around the ducts. Drains were left down to the area and a gastroenterostomy was performed. The patient drained bile for a few days. She lived for almost six months following operation. During this time pain was difficult to control but interestingly enough, she did not develop jaundice until about a month before she died. Thus this method of meeting such an emergency was successful in this case.

FIG. 9. Drawing of restitution of biliary tract to intestine. Actually the jejunum, Roux Y, was employed, not the duodenum as shown.

Comment: This is the first time that such an *in situ* necrosis of the common duct has been seen. It is well known that the blood supply is not over-abundant but with both ends of the duct intact it would seem that it ought to be adequate for preparation of the duct for a distance of 3 to 4 cm. This case further emphasizes the delicacy of these structures.

SUMMARY

We have illustrated some unusual complications of biliary tract surgery. Their avoidance has been pointed out in each case in retrospect.

Emphasis is placed upon the proper incision for the individual, adequate exposure so as to be able to dissect and demonstrate any variation, anomaly, or distortion caused by disease which may be encountered, and upon competent assistance. Although operations upon the biliary tract are very common and are not usually considered serious, errors in technique and management must be avoided in order to preserve the delicate and vital structures, structures which are most difficult to repair unless the injury is recognized at the time of operation.

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NASOGASTRIC INTUBATION: INDICATIONS, COMPLICATIONS, SAFEGUARDS AND ALTERNATE PROCEDURES*

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Postoperative abdominal distension has presented a problem to the surgeon since the first laparotomy was performed. This complication was attacked by the older surgeons with irritating enemas, hot stupes to the abdomen and any other method which offered a hope of relief. Acute dilation of the stomach was not an infrequent postoperative sequela, and was greatly feared in the postoperative period.

John Hunter,[16] in 1790, devised a stomach tube for the administration of stimulants to persons undergoing artificial respiration. He made his tube from a fresh eel skin drawn over a whale probang. From this crude, but ingenious device, all tubes have emanated In 1874, Ewald[6] and Oser,[27] developed stomach tubes of soft, pliable rubber and of smaller caliber than those used previously. Prior to this time, stomach tubes were stiff, hard and only slightly flexible, and produced considerable discomfort when passed. A few soft tubes had been used, but were always inserted with a stylet or mandarin.[29] Ewald[6] and Oser[27] demonstrated that no stylets were needed if the cooperation of the patient could be secured.

In 1921 Levin[21] of New Orleans, developed a rubber tube of small caliber and of sufficient pliability that it could be passed with ease through the nose. This type of tube has, undoubtedly, been used more extensively than any other type of suction or feeding tube. The Levin tube was used with some advantage following abdominal surgery at St. Luke's Hospital, Chicago, prior to 1931. Drainage was by gravity into a bottle at the bedside. It was necessary to irrigate the tube, on occasion, to free it of blood clots and mucous. Although continuous suction was not used, this method prevented accumulation of swallowed air, excessive secretion, persistent vomiting, and acute gastric dilation.

Robertson Ward,[40] of San Francisco, described continuous gastric or duodenal lavage in 1925. It was used as an adjunct to the treatment of general peritonitis, postoperative ileus, intestinal obstruction, and acute gastric dilation. His pioneer work should receive more recognition than it has been given in the past.

In 1931, Wangensteen[38] described his method of continuous nasogastric suction before the Western Surgical Association. He gave as the indications for its use, simple obstruction of the bowel, and postoperative adhesive obstruction. Shortly thereafter Payne and Wangensteen[28] extended the indications for such form of treatment to include patients with postoperative distension, paralytic ileus, and peritonitis. They demonstrated by gas analyses and x-ray studies that a large percentage of the gas which produced distension was swallowed air. Maddock, Bell and Tremaine[25] investigated in animals and humans the phenomena of swallowing air experimentally. They demonstrated that apprehensive patients may ingest enormous amounts of air.

INDICATIONS FOR NASOGASTRIC SUCTION

The beneficial effects of nasogastric suction decompression have been accepted as an important segment of surgical treatment. Each of us knows that it is much more simple to prevent distension of the gastrointestinal tract by suction applied to an indwelling tube, than to relieve a distension already present.

Nasogastric suction is of value in intestinal obstruction, however, there are definite limitations associated with its use. One must be alert to distinguish between simple obstruction and strangulating obstruction. In the former the tube may be definitive treatment, whereas in the latter it is palliative, and definitive surgery is indicated.[18]

Differences of opinion exist as to the routine use of a tube in the postoperative period. Payne

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Presented at the meeting of the Southeastern Surgical Congress, March 10–13, 1958, Baltimore, Maryland.

has stated that nasogastric tubes are indicated in all patients undergoing major abdominal surgery, and particularly in patients having gastrointestinal surgery, or where ileus may be expected after surgery.[30] On the contrary, other surgeons have cautioned against the injudicious use of nasogastric intubation as a routine procedure.[3, 5, 7, 9, 10, 20, 32] It is their opinion that intubation should be reserved for specific indications, rather than practiced as a routine.

Although we do not use nasogastric intubation routinely, it is our practice, on many occasions, to insert a tube for a short period of time, following gastrointestinal and biliary tract surgery. Its purpose is to prevent regurgitation of gastric contents and the accumulation of swallowed air in the stomach. The tube is left in place until the patient has recovered consciousness. The tubes are usually removed prior to the patient's return to his bed from the recovery room. Paralleling the development of nasogastric suction has been the use of the same tubes for feeding purposes. In infants the tubes are usually small catheters and may be introduced through the mouth for each feeding and withdrawn immediately thereafter. In adults and in some infants with pharyngeal paralysis the tube is generally introduced through the nose and left in place for indefinite periods of time. Soft rubber tubes are being replaced by plastic tubes of varying degrees of size and rigidity. Significantly, some of these tubes become hard and even brittle after sterilization or during use, as they come in contact with gastric or duodenal juices. Other tubes contain chemicals to which some patients are acutely sensitive and, as occasionally occurs with intratracheal tubes in the larynx, an acute edema of the nasal, pharyngeal, and esophageal mucosa is seen along the path of the tube. Furthermore, some of the agents used to clean and sterilize the tubes may be responsible directly, or by chemical action, on the tube for this peculiar allergic reaction.

It has been our experience that feeding tubes of soft rubber have been less injurious to mucosal surfaces of the nose, pharyngeal, and esophageal mucosa, than have the plastic tubes.

COMPLICATIONS

Although rare, complications may develop following nasogastric intubation. The following complications have been reported:

(1) Abscess of the nasal septum;[12] (2) otitis media, sinusitis, and hoarseness;[26] (3) ulceration of the larynx;[17] (4) ulceration and stenosis of the esophagus;[13] (5) rupture of esophageal varices;[2] (6) knotting of the tube;[3] (7) inability to withdraw the tube;[18, 24] (8) fluid and electrolyte depletion.[36]

A septal abscess has been reported by Harlowe[12] in a 56-year-old female who experienced pain in the nostril for 5 days, during which time the tube was withdrawn and re-inserted 3 times. A submucosal resection revealed destruction of the cartilage.

Sinusitis and otitis media are not infrequent hazards of the tube.[26] As Wangensteen[39] has noted, children tolerate intubation poorly, and develop otitis media frequently, following intubation. The tube should be well lubricated and the nasopharynx kept moist during intubation, otherwise otitis media may develop from irritation of the mucosa in the region of the Eustachian tube.

In 1946, one of us collected 24 cases of stenosis of the larynx from the literature, and added 4 cases.[13] In each instance a tube had been inserted for feeding purposes or for routine postoperative managements for various periods of time. Tracheotomy was required in 3 patients because of the development of abscesses of the larynx, 4 to 6 weeks following use of the tube. All developed severe chronic laryngeal stenosis

FIG. 1. Lateral x-ray of the neck showing partial destruction of the cricoid cartilage, and postcricoid thickening. The airway is narrowed at the level of the arrow, which is at the cricoid level. These changes followed nasogastric intubation of 17 days' duration. Emergency tracheotomy was necessary 4 weeks later.

FIG. 2. Lateral x-ray of the neck showing partial destruction of the cricoid cartilage, as well as obliteration of the subglottic airway. These changes followed nasogastric intubation of 14 days' duration. Tracheotomy was carried out 6 weeks following intubation.

FIG. 3. Ulcer of lower esophagus in patient with a brain tumor. Tube feedings had been carried out for 21 days prior to death.

(figs. 1 and 2). Frank ulceration of the esophagus may develop from a tube.[37] A patient having a brain tumor, who was subjected to tube feeding for a period of 5 weeks prior to death, serves as an example of this complication. Autopsy revealed an ulcer near the distal end of the esophagus (fig. 3). The scar tissue associated with healing could easily produce a constricting lesion in this area.[24] It is unknown what period of time is required to produce such a lesion. We have seen a stricture form in the distal esophagus following intubation for 8 days.[19] It responded to mercury filled bougie dilation (fig. 4). In our opinion the cardiospasm, seen occasionally following removal of a tube, can be explained on the basis of esophagitis of the lower area, from trauma and irritation of regurgitated gastric secretions.

Esophageal varices may be traumatized by a tube and produce severe hemorrhage. When such pathology is suspected, great caution must be used in ordering the insertion of a tube.

Short and long tubes have been reported as being knotted.[1, 22] In most of the cases the knotting occurs in the stomach, rather than the lower areas of the gastrointestinal tract. In most instances the tubes had been in place for long periods of time, and peristalsis had been reestablished.

Taylor[36] has cautioned that nasogastric tubes may become a dangerous postoperative tool, if electrolytes are removed without adequate replacement. He has shown that suction may remove 180 mEq. (11.02 gm.) of sodium chloride in a 24-hour period. When water is given *ad libitum*, an increased amount of chloride is washed from the gastric mucosa, thus further depleting the electrolytes. Should the tube, purposely or inadvertently reach the duodenum, enormous quantities of alkaline secretions may be lost. This will add to the difficulties of maintaining electrolyte and fluid balance. Moreover, a tube in the duodenum may not prevent gastric dilation or vomiting, which is one of the purposes of inserting a tube.

Other complications include rupture of the mercury filled balloon[2, 24] and partial or complete plugging of the tube and inadvertent tracheal intubation—particularly in the comatose patient.

SAFEGUARDS

In order to prevent the complications of nasogastric intubation, as well as to serve its clinical

FIG. 4 (*Left*). Stenosis of lower esophagus following nasogastric intubation of 7 days' duration. (*Right*) The stenotic area was adequately dilated with mercury filled bougies. Courtesy of F. B. Kelly, Jr.

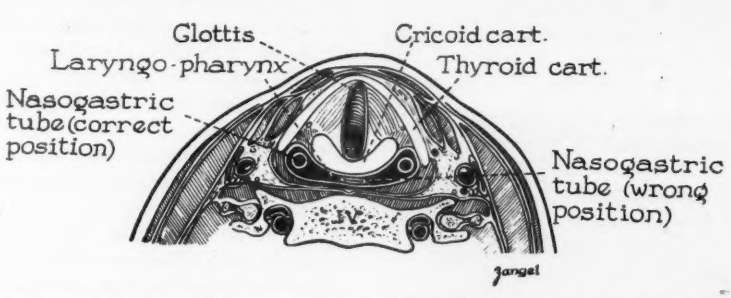

FIG. 5. Drawing of horizontal section of the neck at the level of the 4th cervical vertebra and the cricoid cartilage. Note the compression of the tube in the midline, also the cricoid cartilage in close proximity to the cervical vertebra.

purpose, a tube of proper size, type, and consistency must be selected. The least experienced of house officers are usually assigned the task of inserting a tube. In many instances such an assignment is given prior to proper indoctrination and demonstration. In addition, once the tube is inserted by the Junior House Officer, the further management is relegated to the nursing staff. The surgeon must see that the tube is properly managed while it is in use.

Critical testing of tubes and balloons must be done prior to use. Should a long duodenal tube be selected, the stomach must be emptied prior to installation. Once the long tube has reached the desired area, the balloon should be deflated. This will prevent peristalsis attempting to move it past the obstruction, with the attendant dangers of knotting, perforation, and necrosis. The balloon tube should never be anchored to the nose, because peristalsis pulls on the tube. Anchoring the tube results in ulceration of the nares.

Complaints of nasal pain, sore throat, hoarseness, or pain in the ears, are signals that the tube should be removed or replaced.[13] The points of pressure throughout the course of the tube are in the nose, where the tube may become wedged between the turbinates and the septum, and the postcricoid area of the laryngopharynx. Tubes should not lie in the midline of the pharynx

(fig. 5). At this point, the upper esophageal pinchcock at the entrance of the esophagus, the part of the inferior pharyngeal constrictors known as the cricopharyngeus muscle, keeps the cricoid cartilage pressed firmly against the cervical spine, in an attempt to keep the lumen closed. The posterior esophageal wall in this area, and the mucosa covering the cricoid cartilage, are extremely thin, and, the thin mucous membrane offers little cushioning for these firm surfaces. The pressure and rubbing action of a midline tube produces ulceration of the postcricoid mucosa (fig. 6). Infection may follow the ulceration into the body of the cricoid cartilage. The constant friction of swallowing, breathing, and talking could increase the pressure of the tube against the mucosa covering the cricoid cartilage. Ulceration is followed by perichondritis, and an abscess with destruction of the cricoid ring. The stenosis or tracheal collapse, which may develop, does not necessarily appear until 4 or 6 weeks following the use of tubes.

Once the type of tube is chosen, it should fulfill the following requirements: (a) proper size, (b) proper consistency, (c) sterile.

Frank and Avola[10] report the use of nasogastric tubes in 7 of 8 patients who developed pseudomembraneous enterocolitis. They have cautioned us that the causative organism may be present on an unsterile tube and may be introduced into the gastrointestinal tract by a contaminated tube.

The tube must be small enough to avoid traumatizing the nasal airway. Furthermore, it should not be too stiff, since perforation of a viscus may occur from ulceration caused by a rigid tube. Several reports have appeared of perforations of a viscus found at autopsy. In these perforations of the esophagus near the cardia, a nasogastric tube had been in place for relatively long periods of time. The tubes had been used to supplement the feeding of patients with bulbar poliomyelitis or other neurological conditions, in which the swallowing function had been lost. Associated disturbance of function of the sphincter at the cardia, probably allowed a free reflex flow of gastric secretions into the distal esophagus, augmenting the trophic ulcer action of the tube.

The greatest abuse of nasogastric intubation develops after a tube is successfully passed. Delay, indecision, wishful thinking, and errors in judgement enter the clinical picture at this time. Definitive surgery is required in mechanical and strangulating obstructions. On many occasions, a false sense of security develops by the very presence of a nasogastric tube when active surgical measures should be instituted. We must appreciate the fact that nasogastric intubation is an adjuvant of definitive surgery in the preoperative and postoperative period. It is not a substitute for necessary surgical intervention. It must be remembered that when a tube reaches the small bowel, accumulated fluids in the stomach must be provided with an outlet. It is possible for a surgeon to order nasogastric suction more to give him a feeling of security than to provide a definitive measure of treatment to the patient.

FIG. 6. Hypopharynx, larynx, and esophagus as viewed from behind. Ulceration of the postcricoid mucosa is marked *A*. The roughened posterior surface of the cricoid cartilage is noted by *B*. These changes followed nasogastric intubation of 5 days' duration.

ALTERNATE PROCEDURES

There are many ingenious methods of emptying the gastrointestinal tract of fluid and gas. A Levin tube inserted through the stomach, duodenum, and into the uppermost portion of the jejunum has been utilized by Gilchrist.[11] The tube is brought out through a small stab wound in the abdominal wall. This procedure is simple, easily carried out, and serves to empty the stomach and small bowel.

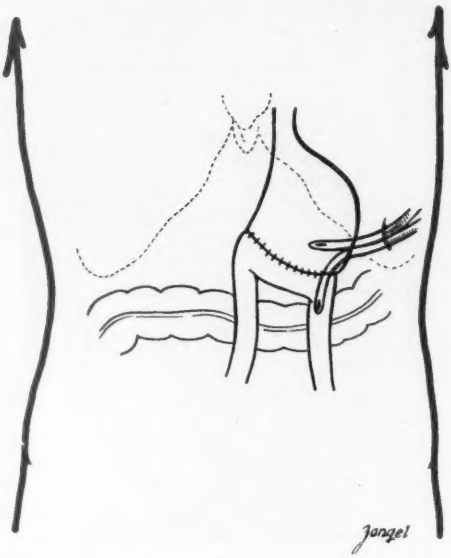

FIG. 7. Gastrostomy with two #14F catheters, one tube projecting across the anastomosis, the other into the stomach. One catheter is used when there is no anastomosis. J. S. Horsley described this method many years ago.

Farris and Smith[8] have utilized, successfully, the Foley catheter as a temporary gastrostomy. We have not used this method in the fear that pressure on the bag of the catheter might produce necrosis of the stomach wall. We have found the two-catheter method of gastrostomy, as described by Horsley,[14, 15] of greatest value. The method is simple, the equipment is readily available, decompression is accomplished, and complications have not developed. The method is of particular value following gastric surgery. Two straight #14F catheters are placed through the anterior gastric wall. The end of one is left in the stomach, the end of the other is threaded through the anastomotic site into the small bowel (fig. 7). The tubes are exteriorized through a small stab wound in the anterior abdominal wall and anchored to the skin The anterior wall of the stomach is attached to the anterior abdominal wall with 3 or 4 chromic absorbable sutures. The stomach is drained by one catheter, while the other is used for feeding the patient if it is desirable. Patients can be kept in nitrogen balance. One small catheter is used when no anastomosis is made. The tubes are removed 7 to 10 days after surgery, and closure is spontaneous. A possible complication of gastrostomy is leakage around the catheter. It is important that the anterior wall of the stomach be attached to the anterior abdominal wall at the point of exit of the catheter. The gastrostomy wound must be made with care, in order to prevent unrecognized hemorrhage. Removal of the gastrostomy tube has posed no problem. After it has served its purpose, it is removed. The fistulas have closed promptly, without sequelae.

We have been interested in surgery of the aged patient for a number of years.[33, 34, 35] More abdominal surgery may be expected in this group of patients as man's life span increases. These people fear nasogastric intubation. A tube prevents adequate respiratory exchange, as well as the removal of mucous from the airways. The irritation of the tube interferes with the patient's rest.

Nasogastric intubation is tolerated poorly in children. Temporary gastrostomy is of particular value in both the extremes of life, childhood and old age.

SUMMARY

Nasogastric intubation has acquired a respected niche in the surgical world. It will undoubtedly be among the significant contributions to surgery of the twentieth century, when such appraisals can be properly documented. Its use has almost eliminated acute dilation of the stomach and the incidence of paralytic ileus is also markedly decreased. It serves a useful role in combatting the distension associated with intestinal obstruction, and is definitive treatment in certain types of bowel obstruction. Furthermore, nasogastric tubes are of significant value for feeding purposes. They are used in patients having esophageal obstruction as well as in the postoperative management of other types of patients.

The circumstances which prompted the development of nasogastric suction are rapidly disappearing, with the antibiotic drugs and early surgery. It is time for us to reevaluate the indications for its use in each patient.

In 1949, Ferguson of Philadelphia, made a plea for "Simplicity in Surgery." He stated that the majority of patients undergoing indicated surgery, and on whom the technical portion of the operation is carried out with due respect for the physiology of the organs involved, and with gentleness to the tissues handled, will get well with relatively few postoperative complications. Ferguson has admonished us to recall the motto of the late Dr. John B. Deaver, "cut well, sew well, get well."

Although it is the exception, serious complications have resulted from nasogastric intubation. It is very important to select a nasogastric tube of proper size, type and consistency. The tube must be inserted with caution, and the patient followed with care.

A valuable alternative method to nasogastric intubation is temporary gastrostomy. It is of particular value in children and in elderly patients.

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AN ENDOCRINE APPROACH TO THYROID CANCER*

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During the past decade as the role of surgery and radiation therapy in the management of cancer has become better defined, there has been increasing emphasis on the biologic characteristics of neoplastic disease. Most of the biological concepts have been derived from empirical as well as experimental clinical observations and considerable amplification has resulted from studies on tumorigenesis in the experimental animal. This knowledge has permitted a "fresh" approach to the understanding of mechanisms of neoplasia as well as to therapy. Two somewhat interrelated concepts have been of particular interest and significance. First, is the elaboration of the role of growth or promoting factors in tumor development[1, 2] and the second is that of tumor progression from initial hyperplasia to invasive autonomous cancer.[3] The first concept implies that development of the neoplastic cell is not necessarily an unexplained change of intrinsic biologic potential but that tumor development may depend upon and be enhanced by normal growth mechanisms. This principle is perhaps best illustrated in experimental tumors of the endocrine system, *viz.*, thyroid, pituitary and breast, where trophic hormones are well known and serve as promoting factors in tumor development. Similar relationships have been established in the laboratory animal between pituitary "growth" hormone and the development of tumors of mesodermal origin.[4] It is not unlikely that there are similar but as yet unidentified trophic influences on cell growth and function in other organ systems.

The second concept implies that a neoplasm is not a sudden transition from normal controlled cellular growth to that of unrestrained growth but that often the phenomenon is a more gradual one, evolutionary in character, occurring in a somewhat heterogenous cell population. Initially, only hyperplasia is seen. However, with continual stimulus and as aided by the processes of mutation and selection, cells develop that have a somewhat different behavior. Most of these cells are still dependent upon promoting or growth factors and consequently the neoplastic changes are still reversible upon withdrawal of these factors. Ultimately, tumors may evolve which are more rapid in their growth as well as anaplastic and invasive. Even in these tumors there is considerable heterogeneity of the cell population and as a consequence variation in biologic potential. As a result, it is not unlikely that following withdrawal of trophic stimuli some of the cells in such a neoplasm undergo complete regression, some exhibit only inhibition of growth, and others continue to progress until the entire tumor seems quite independent and autonomous.

These concepts of tumor development have their greatest application at present in neoplasms involving or arising in the endocrine system, *e.g.*, carcinoma of the breast and carcinoma of the prostate.[5] Although other tumors such as adrenal cortical carcinoma,[6, 7] uterine carcinoma,[8] and thyroid cancer have also been shown to have a somewhat similar relationship to their trophic hormones, there has been little clinical application. The purpose of this paper is to discuss the biologic properties of thyroid cancer in these terms and to suggest how these concepts may be applied in the management of patients with neoplasms of the thyroid.

EVOLUTION OF THYROID NEOPLASMS IN THE EXPERIMENTAL ANIMAL

Considerable insight into the biology of thyroid neoplasms may be gained from studying tumor development in the experimental animal. Through the common denominator of a goitrogen, neoplasms of the thyroid have been produced in several species of animals. Depending on the heredity and strain of the animal, type of goitrogen, presence of an initiating factor or carcinogen, subsequent tumors will vary considerably in their time of development and biologic behavior

* Presented during the Baltimore Assembly of The Southeastern Surgical Congress, March 10–13, 1958, Baltimore, Maryland.

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as well as growth potential. Iodine deficiency, thiourea, and radioiodine alone or in combination have all been productive of thyroid "tumors." An iodine deficient diet in rats has been followed by localized nodular goiters, benign tumors, and frank carcinoma.[9, 10] Similarly, by the administration of a goitrogen alone such as thiouracil, thyroid cancer has been seen in rats after approximately 20 months of treatment. Although metastases have appeared in these animals, all are not independent in their behavior. By prolonging the life and period of stimulation of the experimental tumor with serial transplantation, complete independence of the tumor may be gained. If a carcinogen such as benzopyrene or 2-acetylamino-fluorene or radioiodine is given simultaneously, serving as an initiating factor, the latent period for tumor development is decreased by about one half and the over-all incidence of tumor development is increased.[4] In some species heredity apparently may substitute for the initiating factor since in mice of high tumor strain, "cancer" has followed the administration of a goitrogen alone whereas only "benign tumors" have occurred in animals of low tumor strain.[11]

The role of radioiodine is somewhat more difficult to appraise since its direct action on thyroid parenchyma may be that of a carcinogen and indirectly by virtue of its functional damage upon the gland it may serve as a goitrogen. Thus, is mice the use of radioiodine and thiouracil gave the highest incidence of thyroid cancer.[12, 13] Other species have yielded variable results; some with a high spontaneous tumor incidence have been followed by a lower incidence of tumor production.[14]

Thus, one may demonstrate very nicely both progression of a tumor from an initial state of the reversible hyperplasia to a state of complete independence. In some animals, tumorigenesis apparently may be speeded up by the coincidental administration of chemical carcinogens or ionizing

In studying the development of thyroid tumors in the human it is immediately apparent that conditions similar to those existing in the laboratory animal are seldom duplicated. There are, however, a number of situations which are surprisingly similar and in which there is an increased incidence of thyroid neoplasms.

EVOLUTION OF THYROID NEOPLASM IN MAN

Well differentiated adenocarcinoma of the thyroid is frequently influenced by factors governing normal thyroid growth and function.[15] Other tumors of the thyroid, fortunately less common, which are more poorly differentiated and develop later in life, are much more aggressive in character, and apparently more independent of the previously mentioned trophic stimuli. There is some evidence to suggest that a few thyroid tumors which initially are well differentiated and of low grade malignancy do progress both in their invasive potential and histologic differentiation.

All of the conditions leading to the development of thyroid tumors in animals have in common a persistent stimulus of the thyroid *via* TSH. An analysis of conditions under which thyroid neoplasms in man are known to occur discloses that in many instances there is also evidence for a goitrogenic stimulus.

Iodine deficiency. The relationship of iodine metabolism to diseases of the thyroid is quite complex. In the past most goiters were thought to be secondary to iodine deficiency as evidenced by the decrease in goiter incidence in endemic areas following the addition of iodine to the diet. It is now apparent that there are other factors operative because there remains a fairly high incidence of nodular goiter in the general population. It is also well documented that iodine in excess over long periods of time may in some individuals be productive of goiter and even myxedema.[16] The most convincing evidence for the relationship of iodine deficiency to the development of thyroid cancer in the human is seen in some areas of endemic goiter and iodine deficiency where the incidence of thyroid cancer has been many times its incidence in other parts of the world. Following several decades of iodine administration with reduction of goiter incidence, there was an accompanying reduction in deaths from thyroid cancer to approximately one-tenth of their former figure.[17]

Goitrogenic drugs. Anti-thyroid drugs by blocking the synthesis of thyroxin result in a hyperplastic gland which in the animal may ultimately become neoplastic. Although goitrogenic drugs are employed in long term management of thyrotoxicosis in man, in general a euthyroid state is maintained and the same degree of thyroid stimulation as seen in the experimental animal is not achieved. Thus, there is no comparable long term thyroid hormone deficiency. To date no well documented case of thyroid cancer has been at-

tributed to such therapy. More recently such long term treatment has been employed in children and it is conceivable that any increase in tumor incidence may as yet be unappreciated.

Although the human has not developed carcinoma following goitrogenic drugs, there exist a few individuals in whom there apparently is an inherent biochemical block in thyroid hormone production resulting in goitrous cretins and individuals with varying degrees of hypothyroidism.[18,19] Histologically, the thyroid glands of these patients have shown an intense degree of nodular hyperplasia and in some patients frank cancer with metastases has been present. Somewhat similar pathologic findings have recently been reported in a family with four siblings who were apparently euthyroid. All the siblings have goiters and thyroid cancer was present in two.[20] Minor degrees of hypothyroidism as seen in some forms of chronic thyroiditis also have been associated with an increased incidence of thyroid cancer.[21]

Pituitary stimulation. In keeping with the concept of a growth stimulus as a promoting factor is the observation in patients with thyroid cancer of an increased number of those cells in the anterior hypophysis thought to be responsible for TSH production.[22] Of comparable interest was the reduction in the number of these cells that was apparently brought about by the administration of exogenous thyroid hormone.

Radiation. The role of radiation injury in the development of thyroid cancer is difficult to evaluate. To date there have been no patients developing carcinoma of the thyroid following treatment with radioiodine; external radiation seems to play, if anything, a very minimal role during adult life. Age and growth stimulus apparently are important since there is suggestive evidence that radiation received during infancy may be an important factor in the subsequent development of low-grade thyroid cancer during early childhood.[23] Under such circumstances the radiation may be likened to the initiating factor produced by radiation or a carcinogen in the experimental animal.

Progression. Studies on morphology and iodine collecting function of thyroid tumors in man also suggests a progression from well differentiated functioning tumors to more poorly differentiated and nonfunctioning carcinomas.[24] The most common types of thyroid cancer, namely papillary and alveolar adenocarcinoma in general are quite slow in their growth, in many instances requiring years before causing death of the host. During this time, although there are usually few histologic changes suggestive of a more anaplastic lesion, the tumor does exhibit a more aggressive behavior in terms of local invasion and metastasis,[25] (a situation somewhat comparable to the long standing experimental thyroid tumor of increasing virulence). For the most part these changes occur during a euthyroid state when the tumor is subject to the same trophic stimuli as is the normal thyroid gland. If thyroid cancer is dependent upon TSH, increased growth and more rapid progression should occur in conditions in which there are increased levels of TSH, namely, hypothyroidism and possibly hyperthyroidism. Similarly, conditions leading to a decrease in stimulus to the thyroid might be expected to be followed by regression or inhibition of neoplastic growth. There are well documented observations in man in which both of these seem to be true.

Spontaneously occurring hypothyroidism (with the exception of goitrous cretins and iodine myxedema) is usually associated with either anatomic loss of the gland or loss of its growth and functional stimulus, so that thyroid carcinoma under these circumstances is almost unknown. Four cases, however, have been reported. A sarcoma and a poorly differentiated carcinoma occurred in patients with myxedema of pituitary origin.[26] In the two other patients with an apparently normally functioning pituitary there were no comments as to change in growth characteristics subsequent to the development of myxedema.[27, 28] There are, however, a large number of patients in whom either the hypothyroidism has been induced or the TSH level has been increased by direct administration in which increased tumor growth has been observed. Treatment of thyroid cancer by radioiodine is usually associated with "preparation" of the patient by ablation of normal thyroid function with resulting hypothyroidism. The increase in TSH may be manifest by some increased uptake of radioiodine in about 40 per cent of all tumors.[29] However, such treatment may also increase the size of the tumor, as is well documented by both physical and radiologic examination. Furthermore, there is frequently an increase in symptoms of pain and dyspnea in patients with bony or pulmonary metastases. Although in general, function and growth parallel one another, there may be some tumors which show a relatively greater

growth response and little or no change in function.

There are also patients in whom there has been an associated progression toward a more anaplastic and independent tumor. This type of histologic progression in the human host despite a relatively long residence is unusual but has been reported in a patient with a disease of 15 years duration (initially with hyperthyroidism treated by several incomplete operations, and radium therapy).[30] We have observed one patient in whom progression occurred following treatment of exophthalmic goiter by external irradiation. An additional patient had a mixed type of neoplasm with both well differentiated and anaplastic lesions being present, again suggesting progression.[31, 32]

THYROID CANCER—A DEPENDENT NEOPLASM

These experimental and clinical considerations are in support of the thesis that growth and function of many types of human thyroid cancer may be profoundly influenced by the relative level of thyrotrophic hormone. Consequently, withdrawal of this stimulus may be followed by inhibition or even regression of growth and function (fig. 1). As a corollary, both growth and function are enhanced by elevated levels of thyrotrophic hormone and there is suggestive evidence that progression to a more aggressive anaplastic and independent tumor may be hastened.

CLINICAL APPLICATION

In the application of this concept to the management of thyroid cancer we have had personal experience with 11 patients who have been studied with respect to the dependency of their neoplasm on TSH. Parameters of evaluation have included changes in growth as demonstrated by gross size and radiologic alterations, changes in function as seen by the PBI, and uptake of I^{131} and P^{32} by the thyroid. Such a plan of study has disclosed *regression* of either growth or function in 5 patients; apparent *inhibition* in one patient; and frank *progression* in 4. In

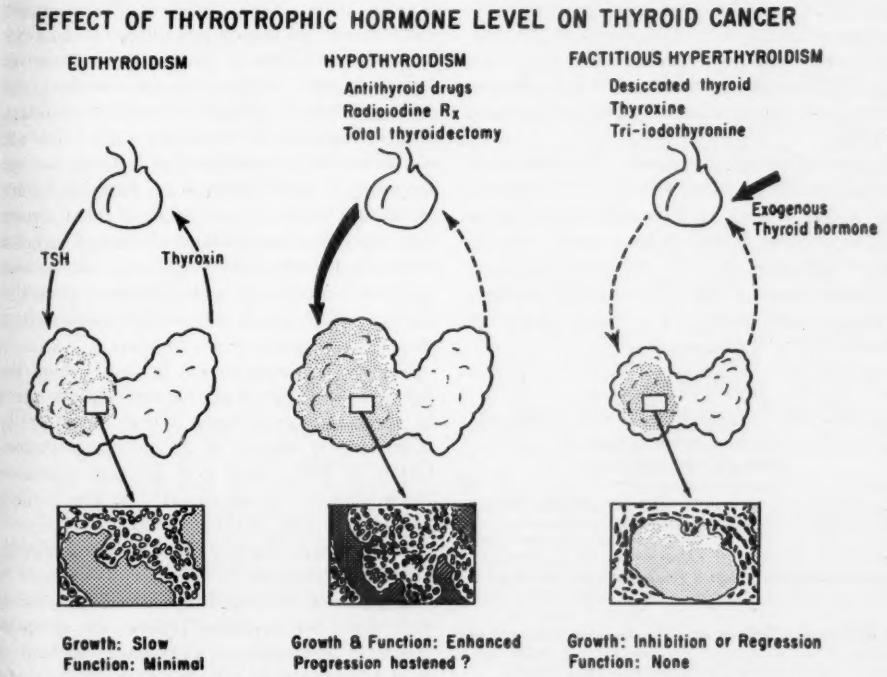

FIG. 1. The servo or feed-back mechanism between the thyroid and the anterior hypophysis results in a varying stimulus to normal and cancerous thyroid tissue depending upon the relative blood levels of thyroid and thyrotrophic hormones.

one tumor there was simultaneous regression and progression of a mixed type of neoplasm. This is thought to be an excellent example of the heterogeneous cell population occurring in a tumor with some elements having progressed to complete independence and others retaining their dependent characteristics.

An examination of the literature discloses a number of patients who have had regression of thyroid cancer following the administration of sufficient exogenous thyroid hormone to depress thyrotrophic hormone output of the hypophysis.[15] Some of these have been most dramatic with recovery from paraplegia, healing of pathological fractures, disappearance of cervical tumors and pulmonary metastases. At times evidence of tumor suppression has been present for as long as 21 years. In others with suppression for as long as 10 years, withdrawal of exogenous thyroid hormone has again been followed by recurrence of neoplastic growth. Judging from the number that are capable of collecting radioiodine, at least 40 per cent of neoplasms have some areas responsive to TSH stimulation. Unfortunately, in many reported cases, because of the varying amounts of thyroid hormone administered as well as ancillary methods of therapy it is difficult to appraise the number of human thyroid tumors that appear to be completely independent of TSH.

In general those tumors with poor differentiation in contrast to those with colloid production have shown little or no dependency upon thyrotrophic hormone. It may be that uptake of radioiodine will prove to be a differentiating characteristic; however, some tumors purely papillary in nature have been shown to regress after withdrawal of growth stimulus.

TABLE 1

Suppression of thyrotrophic hormone in thyroid cancer by exogenous thyroid hormone administration

Thyroid Preparation	Daily Dosage	Criteria of Adequate Dosage	
		P.B.I.	I^{131} uptake
Desiccated thyroid	4–6 gr.	Transient elevation only	Suppressed to below 5%
L-Triiodothyronine	100–200 μg	Falls to hypothyroid ranges (*i.e.*, 1 μg.%)	Suppressed to below 5%

In the management of patients with thyroid cancer sufficient exogenous thyroid hormone must be administered to provide complete suppression of TSH secretion. Since there are no simple and reliable direct measures of TSH levels in the blood, adequacy of suppression is determined by measurement of thyroid function, *viz.*, radioiodine uptake and serum protein bound iodine. Although desiccated thyroid, thyroxin and tri-iodothyronine all inhibit pituitary output of TSH, the T_3 has the distinct advantage of permitting measurement of PBI and its subsequent fall as a criterion of thyroid suppression. All three suppress radioiodine uptake (table 1).

The administration of exogenous thyroid hormone is advised in all patients with a history of thyroid cancer. It is employed therapeutically in those individuals with residual or recurrent tumor when surgical excision is no longer feasible. In those patients in whom there has apparently been complete removal of a thyroid tumor, it is administered prophylactically in an attempt to reduce to a minimum the pituitary growth stimulus for any remaining normal or abnormal thyroid tissue. Cognizance of this principle will also suggest that thyroid hormone be given in those conditions in which the incidence of thyroid cancer is likely to be increased, *e.g.* prolonged administration of antithyroid drugs, congenital goiter with block in thyroid formation, and following I^{131} treatment of hyperthyroidism in younger individuals—until more is known about the neoplastic potential of these glands. Similarly, the administration of thyroid hormone following a subtotal thyroidectomy for nodular goiter will tend to prevent the development of further nodularity, in which differentiation from carcinoma may be most difficult.

Further emphasis should be made upon the danger of any type of goitrogenic stimulus either by way of drugs, induced hypothyroidism with radioiodine or surgery, or by prolonged administration of TSH. Such may not only stimulate the growth of thyroid cancer, but also perhaps influence its rate of progression.

When employing this principle on a prophylactic basis it is our practice to advise three to four grains of desiccated thyroid a day, realizing that there are no good criteria for adequate pituitary suppression. Desiccated thyroid is used here because of its low cost. When therapeutic action is desired and a criterion of ade-

quate pituitary suppression is needed, tri-iodothyronine is employed in gradually increasing doses—utilizing fall in protein bound iodine and depression of radioiodine uptake in conjunction with the development of mild hyperthyroidism as criteria of adequate treatment. Fifty micrograms of tri-iodothyronine is administered in divided doses daily for ten days to two weeks. If there are no symptoms suggestive of hyperthyroidism, the dosage is gradually increased to 100 μg. and subsequently to 150 or 200 μg. daily.

In general the dose has been increased until there are some symptoms of hyperthyroidism and then the dosage has been slightly reduced. It has been our experience that there is a tremendous variability in tolerance with practically all patients capable of ingesting 100 μg. daily and some taking as high as 300 μg. daily with no symptoms of hyperthyroidism. A few apparently have had some accumulative effect since while initially there were no symptoms of hyperthyroidism on 150 μg. daily these gradually developed but were lessened by reducing the dosage to 100 μg. daily.

Some of the equivocal results in the past may have been on the basis of inadequate thyroid hormone administration. In animals a goitrogen may still give its effect unless adequate doses of thyroid hormone are given concurrently. This is also true in a few ptients with thyroid cancer in whom an initial inadequate dosage was accompanied by continued tumor growth, whereas following an increase in the amount of desiccated thyroid administered, tumor regression resulted.

It is also apparent that decrease in size of a nodule in the thyroid after thyroid hormone administration cannot be used as a criterion of benignancy but merely that of dependency. Furthermore, the role of this concept must be weighed in evaluating other methods of treatment of thyroid cancer. Radioiodine treatment has been cited as being responsible for an apparent increase in survival rates in patients with thyroid cancer.[33] It is not unlikely that some of the beneficial results attributed to radioiodine may as well be the consequence of exogenous thyroid hormone, since most of the patients following ablation of normal thyroid tissue are given some form of thyroid hormone in order to maintain a euthyroid state.

SUMMARY

The biologic behavior of certain types of thyroid cancer suggest that the trophic hormones for normal thyroid growth and function may also serve as promoting factors in the development of experimental as well as human thyroid cancer. Implicit in such a concept is reversibility of neoplastic growth because of its dependence upon TSH and progression of growth with continual stimulation. Such a concept has direct application in: (1) the management of inoperable thyroid cancer; (2) prophylaxis against recurrence following operations for thyroid tumors; (3) prophylaxis in those conditions in which there is an increased incidence of thyroid cancer and in the potentially recurrent goiter; and (4) the evaluation of the treatment of thyroid cancer by radioiodine.

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OXYPHILIC GRANULAR CELL ADENOMA OF THE PAROTID GLAND: REPORT OF A CASE

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Oxyphilic granular cell adenoma (oncocytoma) is a rare tumor occurring most often in the parotid gland and considered by most persons to be a benign lesion.

The characteristic cells of this tumor have been described by Hamperl who called them "oncocytes." They are epithelial in origin, larger than the normal cells of the parotid gland and have a granular or reticulated cytoplasm. Their nuclei are dark-staining, irregular in contour and often pyknotic. They are frequently arranged in columns or strands.

Hamperl[1] found these cells in uvular, sublingual and submaxillary glands. He believed that they seldom occur in people under 50 years of age, but occur commonly in people between 50 and 60 years and in almost all individuals beyond the age of 70. At times, these cells form areas of hyperplasia disposed as solid or adenomatous foci. Oxyphilic cells have been found in all of the salivary glands, the thyroid, parathyroid, hypophysis, testis, Fallopian tubes, liver, stomach and the glands of the pharynx, trachea and esophagus. Nohteri[2] has confirmed the findings of Hamperl. Skorpil[3] found five instances of nodular areas of hyperplasia consisting of oncocytes in 100 salivary glands. Luis Meza-Chavez[4] studied normal parotid glands in 51 patients and found oxyphilic cells in 90 of these. Stout[5] found such cells in the bronchi of adults. Oncocytes may be multicentric in origin, a finding of importance when considering recurrence of this tumor.

McFarland[6] reported what seems to be the first pure adenoma of the parotid gland composed of oxyphilic granular cells. At first, he doubted whether this was a true adenoma. The tumor recurred 10 years later. McFarland,[7] in a later review of mixed tumors of the salivary gland, removed it from this group and called it an adenoma. It was the only example of oxyphilic granular cell adenoma which he found in 297 mixed tumors reviewed. Wheelock and Madden[8] found no such lesion in 13 patients with uncommon tumors of the salivary glands. Ackerman[9] accepted only 8 reported examples of this growth as being true oncocytomas. Rawson, Royster and Horn[10] found no oxyphilic adenomyomas in 160 patients with tumors of the salivary glands. Foote and Fresell[11] list one instance in 800 tumors of the major salivary glands reviewed by them. Bauer and Bauer[12] reported 5 patients with this neoplasm in a study of 143 cases of neoplasm of the salivary glands. The only reference to the possibility of these lesions being malignant was presented in their paper. Most observers believe oncocytomas are benign although recurrences have been reported. The patient reported by Boley and Robinson[13] is the only discovered report of a bilateral oxyphilic adenoma. An incomplete survey of the literature discloses 28 reports of patients having oxyphilic granular cell adenomas.

The clinical features of this growth are minimal. The presence of a swelling or tumor is the commonest complaint. It is most apt to be present in the parotid gland. Pain or discomfort is not often present. Increase in size occurs slowly and the growths are not often large or disfiguring. The patient is usually beyond 50 years of age. Systemic symptoms do not occur. There are no significant clinical laboratory findings. Pathologic studies reveal the typical adenoma with the characteristic oxyphilic granular cells. There is an excess of lymphocytes not localized into follicles. Surgical removal is the treatment of choice and recurrence is uncommon.

CASE REPORT

W. M., a 77-year-old white married male was admitted to Misericordia Hospital, Philadelphia, on November 27, 1956. He complained of a lump which had been on his face (in front of the left ear) for 3 to 4 years. It had been slowly increasing in size. The mass did not pain him, but was tender

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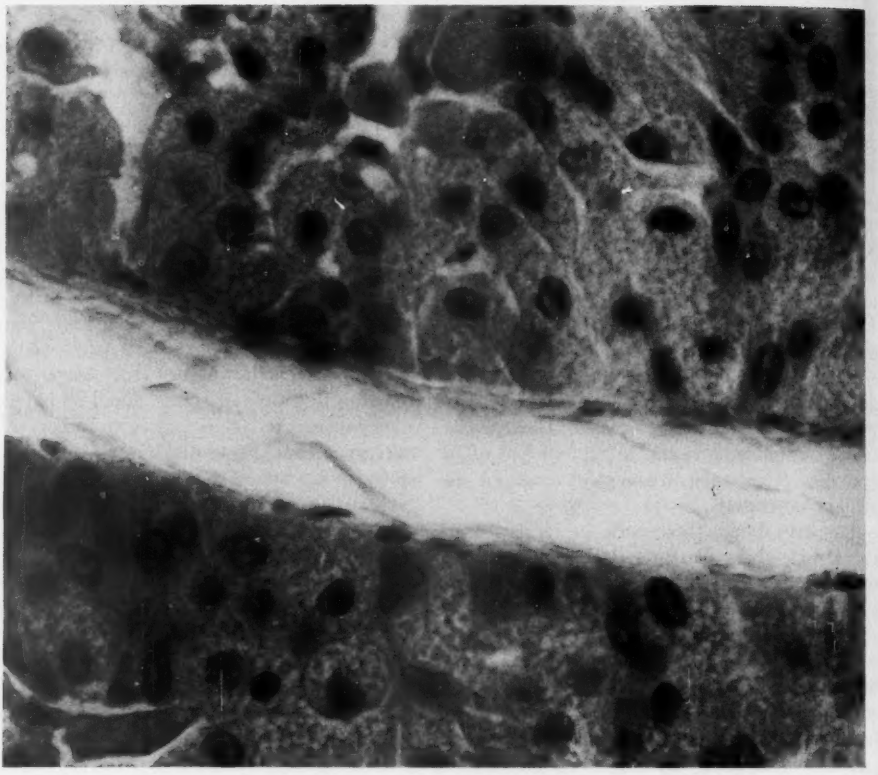

FIG. 1. Photomicrograph of oxyphilic adenoma showing typical granular cells and cordlike arrangement. × 350

to the touch when shaving or if he accidentally struck his face. There were no other complaints. His past medical, family and social history were entirely negative.

Physical examination revealed a moderate hypertension, his systolic blood pressure being 178 mm. Hg and the diastolic 100 mm. In the region of the left parotid gland, just anterior to the ear, there was an ovoid, firm, freely movable tumor about 3 by 2 cm. in size; it was not tender. There was a slight enlargement of the area of cardiac dullness and a Grade I systolic murmur. A bilateral inguinal hernia was present. Physical examination was otherwise normal. All laboratory findings were normal, as was an x-ray examination of the chest.

The patient was operated upon on November 28, 1956. Exploration disclosed a firm, purplish, ovoid tumor about 3 by 2 cm. in size arising in the superficial part of the upper left parotid gland. It was well circumscribed. The growth was completely excised and the incision closed. His recovery was uneventful and the wound healed without infection. The patient is living, well and without recurrence at the present time.

DISCUSSION

The pathologist's report of the examination of the tumor is as follows: "Sections of the lesion of the parotid gland show a well encapsulated tumor, consisting of large eosinophilic cells with abundant cytoplasm and rather small regular vesicular nuclei. They are arranged in cords and in some instances they are grouped around open spaces, reminiscent of glandular formation. There are, however, no true glands seen. The cells are very uniform in appearance and do not exhibit malignant tendencies. There are ductlike structures also seen within the mass of the tumor. I believe that this tumor represents a very infrequently encountered benign tumor of the parotid called an oxyphil adenoma."

SUMMARY

In conclusion, a report of a patient who had a successful removal of an oxyphilic granular cell adenoma of the parotid gland is presented together with a brief description of the pathological and clinical findings. The adenoma has not recurred 13 months after its removal.

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SURGICAL TREATMENT OF DIVERTICULITIS*

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INTRODUCTION

Any patient with diverticulitis may develop one or more of three serious complications of the disease. Obstruction of the large bowel may occur, the colon may perforate, or a fistula may develop between the colon and the bladder. In women, a fistula may form between the colon and the vagina. These patients must be operated upon as without expert surgical care, they may not survive the acute abdominal emergency. Many of these patients are obese and over 60 years of age. Less than twenty years ago, the mortality rate for the surgical treatment of diverticulitis was as high as 50 per cent.

The mortality rate has been strikingly reduced in recent years. One hundred and thirty-one patients with diverticulitis have been operated on at the clinic during the last ten years, with a mortality rate of 1.5 per cent. Since 1950, 102 patients have been operated upon without a death. On the other hand, the morbidity in terms of operative procedures, complications and length of hospitalization is still high. Some of these patients are disabled for months. They often require two or three operative procedures and the total cost of their medical and nursing care is a tremendous financial burden for any family. It is time that we reviewed the surgical management of diverticulitis to determine how this morbidity can be reduced.

DIVERTICULITIS WITH COMPLICATIONS

Surgical principle. If the patient has complete obstruction of the colon, or a large inflammatory mass surrounding a perforation, or a colovesical fistula, he should have a proximal colostomy. He also needs incision and drainage of an abscess, if one is present. Any attempt at resection of the lesion under these conditions is hazardous. Not only is the life of the patient endangered, but further complications, such as residual abscesses or fistulas, are likely to result.

Inadequate surgery such as local operative procedures on the colon also add to the complications associated with diverticulitis.

Drainage of the abscess alone, without a colostomy, usually will result in a colocutaneous fistula. Direct suture of a perforation without a proximal colostomy frequently results in a persistent fistula. If a colocutaneous fistula is already present, an attempt to excise the fistulous tract and close the colonic opening will probably lead to a second fistula. Not only has the patient made no progress, but the inflammatory reaction surrounding the sigmoid colon has been increased. The resection of the sigmoid which will be required before the patient is cured has been made more difficult. The same is true when a direct attack is made on a large colovesical fistula.

With a proximal colostomy, the acute inflammatory process gradually subsides. A colovesical or colocutaneous fistula may heal. This should not mislead the surgeon into closing the colostomy without resecting the diseased bowel. Closure of the colostomy without resection frequently results in recurrence of symptoms or the development of more complications, such as a second perforation or obstruction.

How soon the resection of the colon should follow the establishment of the colostomy will depend upon whether or not carcinoma has been ruled out. The possibility that the lesion in the sigmoid may be malignant and not inflammatory must be considered. Indeed, both carcinoma and diverticulitis may be present. Almost all diverticulitis occurs in the sigmoid colon. The sigmoid is also the most common site for carcinoma of the colon. They occur in the same age group. The symptoms may be identical. Each may lead to obstruction, and each may perforate or result in a fistula. We rely largely upon the barium enema examination to distinguish between carcinoma of the sigmoid and diverticulitis. Yet in 20 to 30 per cent of patients with diverticulitis, carcinoma cannot be excluded by the barium

* Presented during the Baltimore Assembly of The Southeastern Surgical Congress, March 10–13, 1958, Baltimore, Maryland.

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enema. If the surgeon cannot be sure that the lesion is not malignant, he must proceed with resection and anastomosis within 2 to 3 weeks regardless of the extent of the inflammatory reaction present. Under these circumstances the resection is likely to be difficult. All of the surrounding structures, including the left ureter, will be densely adherent to the mass. Because the operation has been carried out in an infected field, localized peritonitis, wound infection or residual abscess may occur. The 2 deaths in this group of 131 patients occurred in cases of this type. These unfavorable operating conditions must be accepted. The mistake of assuming that a carcinoma of the sigmoid is diverticulitis may cost the patient his life.

On the other hand, if carcinoma can be excluded, resection should be delayed for at least 4 months, and preferably 6 months. Acute inflammation, even small abscesses, are frequently still present 3 months after a proximal colostomy has been established. In a few patients the acute inflammatory reaction will not have entirely subsided in 5 or 6 months. This is most likely to be true when a large abscess cavity does not have adequate drainage. If the operating conditions are satisfactory, the colostomy may be closed at the time of resection. This is often possible when the resection is carried out after the inflammatory reaction has completely subsided. This eliminates a third operative procedure, closure of the colostomy. It should not be done, however, if the surgeon is concerned about the anastomosis. With the protection of a proximal colostomy, a small defect in the suture line will heal. Without the colostomy, it may lead to an abscess, and eventually a fistula.

With this staged operative attack, properly timed, an elderly, obese patient can survive a potentially fatal complication of diverticulitis. It is largely responsible for the greatly reduced mortality associated with the surgical treatment of this disease. It does mean two and possibly three operative procedures. Any attempt to avoid a proximal colostomy in such a patient is dangerous. It may lead to more rather than less surgery. We frequently see patients who have had four or five unsuccessful operative procedures to correct a perforation of the colon due to diverticulitis. These patients still have to have the staged operative procedure which they should have had in the first place.

Technique. A few points in technique are important. (1) The colostomy is established in the right or proximal portion of the transverse colon. Many of these patients require mobilization of the splenic flexure at the time of resection. A colostomy in the mid or distal transverse colon may interfere with an adequate resection at the second operation. (2) The bowel should be completely divided so that no fecal material reaches the area of inflammation. (3) If a large abscess is present, it should be drained. The surrounding inflammatory reaction will subside more quickly. (4) The resection will be easier and the patient's convalescence smoother if the inflammatory reaction is given an adequate time to subside. (5) The bowel removed should include most, if not all, of the diverticula-bearing colon. This not only reduces the risk of recurrence of the diverticulitis, but it permits a more secure anastomosis. The walls of the diverticula are very thin and are easily torn by the suture. If many diverticula are present at the suture line, it is apt to leak. For this reason, it is frequently necessary to mobilize the splenic flexure and to remove most of the descending colon. (6) The amount of mesentery that should be removed will depend upon whether or not carcinoma has been excluded. If the lesion is definitely inflammatory, the mesentery can be divided close to the bowel and the inferior mesenteric artery preserved. This insures an adequate blood supply to the anastomosis. It reduces the chance of injury to the ureter. On the other hand, this type of resection is a serious error if the lesion should prove to be malignant. Even though the surgeon subsequently removes the remaining mesentery, the dissection has been through, rather than around the lymphatics draining the tumor. Malignant cells may now be scattered throughout the operative field. This greatly increases the danger of a local recurrence of the carcinoma. If carcinoma has not been ruled out by the history or by the barium enema study prior to operation, the lesion should be carefully examined before the mesentery is divided. After the sigmoid has been mobilized, it may be possible to depress the lesion far enough into the pelvis so that the mucosal surface can be felt by a finger introduced into the rectum. The firm, raised margin of a carcinoma is easily recognized. If the mucosal surface is smooth, it is probably diverticulitis. If the surgeon still cannot rule out malignant

disease, he must carry out a cancer-type of operation despite the unfavorable operating conditions.

DIVERTICULITIS WITHOUT COMPLICATIONS

For years, surgery has been limited to the treatment of the complications of diverticulitis, that is, obstruction, perforation or a fistula. If we wait until these complications develop, multiple operative procedures and prolonged surgical and nursing care are inevitable. These complications can develop without warning. When they do, the surgeon has no alternative. Most patients with diverticulitis, however, have had repeated attacks of acute inflammation over a period of months or years. Some have spent many weeks in bed, at home or in the hospital, waiting for the inflammation to subside. Finally an attack occurs which does not subside, or it begins with a sudden, severe pain in the left lower quadrant, caused by a perforation of the colon.

Although they may be improving so far as symptoms are concerned, some of these patients will be found to have a significantly narrowed sigmoid colon on barium enema examination. The edema associated with the next bout of diverticulitis could produce an acute surgical emergency—complete obstruction of the large bowel.

Other patients with diverticulitis develop urinary frequency or lower abdominal distress on urination. The significance of these symptoms may not be appreciated even by the physician. This is frequently followed by the passage of gas and fecal material per urethra. The patient now has a colovesical fistula.

It seems obvious that gastroenterologists and surgeons should revise their indications for surgery in this disease. Patients with recurrent attacks of diverticulitis, especially if these attacks are increasing in severity, patients with marked narrowing of the lumen of the colon, and patients with diverticulitis who develop dysuria should be advised to have a resection of the diseased bowel. If this is done before perforation, obstruction or a fistula develops, these patients can have a one-stage resection of the colon with a primary anastomosis. They can be operated upon with a very low mortality rate and with few serious complications. They are relieved of their recurrent attacks of diverticulitis. They avoid the serious and possibly fatal consequences of a perforation or obstruction of their colon.

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THE MANAGEMENT OF THERMAL BURNS*

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The problem of burns has been an increasing one. In spite of recent improvements in care and knowledge concerning systemic manifestations, there remains a very high morbidity and mortality. This is evidenced by the fact that the hospital course of burned patients has been shortened little except where interest in treatment has been stimulated by the combined efforts of groups concerned with such problems. The over-all reports are not convincing that morbidity and mortality have been appreciably altered during the past decade. Public health statistics[23] indicate a constant number of deaths from burns over a five-year period. Approximately 6500 patients die each year from the effects of burns.

The incidence of burns seems ever-increasing, although great efforts have been made by many[5, 19] to bring to the public the necessity for preventive measures. Industry and high speed mechanization continue to furnish a large number of what should be preventable lesions. Statistical reports, however, indicate that the vast majority of accidents occur in and around the home in non-occupational pursuits. In a recent survey of burned patients treated at Emory University Hospital during the past seven years, it was found that 75 per cent were non-occupational injuries. Over 80 per cent of the cases were caused by carelessness on the part of the burned subject or his guardian.

PHYSIOLOGIC EFFECTS

The physiologic effects following a major burn are those of adaptation to any injury, plus the specific damages of the burn. As seen in table 1, an intense stress situation such as a major burn, results in profound metabolic changes.[18, 20] There is increased gluconeogenesis and glycogen utilization and increased protein breakdown, giving rise to deficits of these substances. Oxidation of fat is slower and this is accompanied by impairment of the processes of synthesis, leading to an over-all negative metabolic imbalance. Furthermore, there are increased losses of nitrogen and sugar in urine and feces, thus adding to the imbalance. These changes are regarded as "obligatory" and apparently persist for a two to three week period.

As indicated in table 2, the foremost physiologic effect of a major burn is the "sequestration" of large amounts of serum in the wounds immediately after burning, resulting in decreased blood volume, hemoconcentration, and lowered blood pressure.[7] In addition, there is increased erythrocyte fragility, with resulting hemolysis[3, 18] plus loss of protein, mainly albumin, through the wounds, giving rise to red cell and protein deficits. This is followed in three to five days by resorption of the fluid and electrolyte sequestrum, accompanied by diuresis, if recovery is to ensue.

There is, then, a gradual return to relatively normal metabolic balance. When renal excretion of catabolic products lessens to the point where it is proportionate to intake, it is the end point of negative metabolic imbalance. But, in addition to these metabolic effects, there are continuing losses of proteins, electrolytes, and blood from the burn site. These cease only when re-epithelization is complete. It must be remembered that infection, dressing changes, and operative procedures are continuing stimuli to the stress response; and variations from positive to negative imbalance cease only when the burn is healed.

Pathologically, the effects of burns are partial destruction or damage to a vital organ—the skin. Various methods have been devised to distinguish partial thickness from full thickness burns, but this distinction is yet made largely by clinical evaluation. Loss of parts of the skin results in partial loss of a vital temperature-regulating mechanism, the principal barrier against bacterial invasion, and the protection to underlying structures, without which survival is impossible. Fatality or survival to a large extent depends on the percentage of skin loss.

* Presented during the Baltimore Assembly of The Southeastern Surgical Congress, March 10–13, 1958, Baltimore, Maryland.

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TABLE 1

General response to injury

1. Increased epinephrine secretion (transient) vasoconstriction, bronchodilatation, increased cardiac output
2. Increased adrenal steroid secretion
3. Increased rate of nitrogen metabolism
4. Renal loss of nitrogen
5. Accelerated gluconeogenesis
6. Increased glycogenolysis with elevated blood sugar and increased renal loss of sugar
7. Increased oxidation of fats
8. Potassium loss
9. Initial retention of N_a and H_2O, followed by diuresis

TABLE 2

Specific response to burns

1. Increased capillary permeability
2. Loss of serum—electrolytes and proteins—into wound
3. "Sequestration" of H_2O and N_a in edema fluid
4. Subsequent mobilization of "sequestrum" with diuresis
5. Decrease in red cell mass caused by sludging and hemolysis, and "sequestration"
6. Direct loss of H_2O, electrolytes, proteins, and blood from wound
7. Repeated trauma (fever, dressing changes, operations) further stimulate general stress response and magnify losses of blood and proteins

TABLE 3

Estimation of fluid requirements

1. EVANS' formula:
   (1 cc. colloid / 1 cc. electrolyte solution) × % of burn × weight in kg.
   Plus 2000 cc. water
   A. Give above in first 24 hr.
   B. Give half of first day's requirements in first 8 hr.
   C. Treat burn over 50% as only 50%
   D. Second day's requirements approximately half of above
2. After first 48 hr.
   A. Electrolyte requirements minimal
   B. Dehydration may occur
   C. Continuing direct blood and protein losses best replaced parenterally

TREATMENT

The treatment of burns is directed towards minimizing the effects of altered physiology and restoration of the skin in as short a time as possible. The early problem in management is that of preventing shock and replacing fluid and electrolyte losses. Pioneer work by Cope and Moore[7] and other investigators culminated in the efforts of Evans and associates[10] to formulate a guide for adequate replacement. This formula takes into account both surface area involved and the size of the patient.

Experience has shown that in some instances, particularly in second degree burns, the amount of colloid substance required is slightly excessive. Hence, the formula has been modified by Reiss and associates[21] to provide an excess of electrolyte solution over colloid. The rate at which fluid, electrolytes, and plasma are lost, following a burn, is represented by a parabolic curve with its peak occurring during the first 8 hours. Thus, it is recommended that one half of the first day's requirement be given during the first eight hours following the burn, and that two-thirds the calculated requirement for the first forty-eight hours be given during the first day. It is recommended that burns involving more than 50 per cent of the body surface be treated as only 50 per cent burns.

The exact colloid requirement is questionable. Although some have advocated the exclusive use of whole blood, it is believed that in many instances, plasma, or plasma volume expanders, can be used equally satisfactorily. This is particularly true of those burns which are second degree. When the burn is third degree, the use of whole blood is necessary to prevent a deficit of red blood cell volume. Periodic hematocrit observations are the best clinical guide as to whether whole blood is required, the goal being to keep the hematocrit between 40 and 50.

The electrolyte solution generally used is physiologic saline, but Artz has emphasized that this solution is hypertonic to plasma for the chloride component. Hence, large infusions of normal saline tend to produce hyperchloremia. He advocates the use of lactated Ringer's solution as being a more nearly balanced electrolyte solution.[21]

It must be emphasized that any formula serves only as a guide for estimation of a given patient's requirements. Clinical signs of adequate urinary output (30 to 60 cc. an hour), normal hematocrit, stability of the pulse rate, and blood pressure,

and absence of disturbed sensorium give the final answer as to whether replacement therapy is satisfactory.

After the first 48 hours, fluid requirement is drastically reduced; and the need is often met by oral intake. The rate of loss is markedly lowered; and as diuresis begins and the fluid and electrolyte sequestrum is mobilized, the vascular compartment becomes saturated. Ill-advised attempts to match urinary output with parenteral intake, particularly with electrolyte-containing fluids, may result in pulmonary edema. Hence, intake is preferably reduced to an amount sufficient only to prevent dehydration. This is assured when serum sodium is kept at an approximately normal level.

Losses of red blood cells and serum proteins from the wound continue until skin is replaced. In addition, the combination of anorexia plus catabolic loss of fat, carbohydrate, and protein results in a cumulative nutritional loss. Blocker and others[1] have advocated early and prolonged force feeding of a high protein liquid diet by tube. Evans and Butterfield[9] have described a condition characterized by massive increase in glycogen breakdown leading to hyperglycemia and glycosuria. This is associated with a very poor prognosis and is accentuated by high intakes of carbohydrates. On this basis, forced feeding is perhaps contra-indicated. Although Blocker and associates[2] have shown by S-35 labeled methionine uptake studies that anabolism in burned patients proceeds at a normal or increased rate, the work of Moore and Ball[18] indicates that there is a phase of nitrogen rejection after severe injury which may last as long as two weeks. During this time, increasing oral nitrogen intake is reflected largely by increased excretion. Nevertheless, it would seem wise to provide an excess of calories and proteins so as to permit as prompt positive metabolism as possible. In addition, during the phase of nitrogen rejection, direct replacement of blood cells and serum proteins can be accomplished by both whole and fraction blood transfusions. This should be continued as needed to maintain a hematocrit of approximately 45 and normal levels of serum proteins throughout the period of treatment.

In this connection, the use and care of available superficial veins becomes a matter of considerable importance; and venipuncture and phlebotomy should be entrusted to the more capable house officers. Unwise selection of a site for phlebotomy or development of superficial thrombophlebitis can be disastrous to proper care of the patient.

Efforts to assist re-establishment of physiologic equilibrium by giving ACTH and cortisone have been shown to be of no value except is states of adrenal exhaustion.[16] Steroid therapy was first thought to be beneficial for many burn problems, but it has failed to measure up to original anticipations. Results of clinical trials show that administration of these hormones does not materially alter the course of burned patients. The process of healing is unaffected, and the problem of infection is not materially lessened; possibly, it is even made worse since steroids may depress the defenses against infection. There is some euphoria and apparent temporary improvement following the use of these substances in chronically debilitated patients. The tendency of these hormones is to produce increases in nitrogen excretion and hence increase the nutritional deficit. It is not recommended, therefore, that burned patients be treated with ACTH or cortisone except in instances of true adrenal insufficiency.

Hypothermia has been considered as a possible means of lowering tissue metabolic activities during the early phase of a burn and thus lessening systemic effects. Martin, Stone, and Cooper[17] have shown that electrolyte disturbances can be lessened during the immediate period following experimentally produced burns in the dog and that there is no delay in the healing processes following hypothermia. The rate of fluid loss was significantly diminished. Although clinical application of these data has not been made, hypothermia may prove to be of value in those individuals with such extensive burns as to have severe changes in the metabolic functions and fluid and electrolyte losses. It has also been suggested that hypothermia may facilitate more extensive excision and grafting by reducing blood loss and increased metabolism at this time.

When the patient has survived the period of burn shock, the problem then becomes one of removal of destroyed tissue and replacement of skin by grafting. Both chemical and mechanical means have been advocated to accelerate the removal of the burn eschar. Various proteolytic enzymes of both human and bacteriologic derivation have been used topically and parenterally, with no convincing evidence that these offer any advantages over simple mechanical cleansing.

Surgical excision of the burned area has a defi-

TABLE 4

Complications of burns

1. Early
   - A. Pulmonary edema
   - B. Pneumonia
   - C. Transfusion reactions
   - D. Lower nephron nephrosis
2. Intermediate
   - A. Severe local infections
   - B. Septicemia
   - C. Thrombophlebitis
   - D. Adrenal exhaustion
   - E. Urinary tract infections
   - F. Curling's ulcer
3. Late
   - A. Severe nutritional depletion
   - B. Contractures and keloid
   - C. Homologous serum jaundice
   - D. Urinary calculi

nite usefulness in small deep burns. In larger burns, the patient's tolerance for extensive excision of burned skin is compromised by the effects of the burn, and the indications are thus sharply limited. In addition, clinical distinction between second degree and third degree burns in borderline cases is inaccurate. A convenient means of accurately determining the depth of a burn would lead to a wider application of early surgical excision.

Until final skin coverage is obtained, various complications, both directly and indirectly related to the burn, may arise (table 4). Respiratory complications are frequent in the early phase of the post-burn period. Deep burns of the head and neck, inhalation of smoke and noxious gasses in burning buildings, and pre-existing pulmonary disease are particularly prone to result in these complications. The use of tracheostomy in the treatment of burns has been life-saving in many such instances. Excessive secretions can be more adequately handled by nursing personnel and adequate oxygenation is permitted. Tracheostomy also facilitates the administration of anesthesia for operative procedures. It is important that an airway be established from the beginning, rather than waiting until the patient is in trouble. It is easier to perform a tracheostomy before edema develops in massive burns of the head and neck area.

Later in the course of burn treatment, the foremost complication is that of infection, both local and systemic. The break in the skin barrier provides an easy pathway for invasion of adjacent areas by a multiplicity of organisms, and local infection is an almost inevitable accompaniment of full thickness skin loss. Septicemia is a major complication which probably occurs much more frequently than realized and accounts for a high percentage of deaths.[15] Antibiotics, topically and systemically administered, have not solved this problem. The variety of possible organisms is so great and the avenue of invasion so broad that frequently a few susceptible organisms may be excluded, only to be followed by overwhelming invasion by organisms insensitive to available antibiotics. Because of the likelihood of development of resistant strains, it would seem wise to withhold the broad spectrum antibiotics from "prophylactic" or empirical use until a specific indication determined by culture and sensitivity studies arises.

Topical application of various antibiotic ointments, sprays, and impregnated dressings has been widely recommended and is not entirely valueless. There are no substitutes, however, for frequent mechanical cleansing of the burned area.

Superficial thrombophlebitis is a frequent nuisance complication and may at times assume serious proportions. This is probably the effect of three factors: the presence of adjacent infection, the introduction of foreign materials into the veins at the site of phlebotomy, and the increased viscosity of blood during the early burn period. There is some indication that an anti-heparin substance may be released as a result of burns. Deep thrombophlebitis is recognized much less frequently, but does occur with all the attendant problems of possible pulmonary embolus and chronic post-thrombophlebitic syndrome of the legs.

Urinary tract infections are frequent. Indwelling catheters should have careful attention to prevent the accumulation of debris, and they should be changed frequently. Urinary calculi also occur frequently, probably as the combined result of infection and increased excretion of calcium during the prolonged immobilization that accompanies massive dressings and operative procedures.

Other complications directly related to the burn may be regarded as effects of the normal physiologic response. Among these are Curling's ulcer, adrenal exhaustion, and liver failure. The chain of events leading to increased viscosity and

stasis of blood flow with mucosal congestion and stagnant anoxia of mucosa[11] provides a fertile medium for the increased hydrochloric acid secretion that accompanies elevated blood levels of steroid hormones.[12] The exact incidence of Curling's ulcer is not ascertained, but reported mortalities in most series of burns include one or more fatalities caused by complications of this ulcer.

The normal patient seems to have enormous capacity for adrenal response; but in extremes of life, patients already debilitated by pre-existing disease, and the increasing number of patients who have previously had steroid therapy for other disease, states of adrenal exhaustion with Addisonian crisis may occur.

The cumulative effects of shock with decreased hepatic circulation, altered metabolism, and plasma and multiple blood transfusions, results in a significant incidence of liver dysfunction and actual hepatitis[6, 14] and contributes other instances of fatalities caused by complications of burns.

The ultimate goal in treatment of a burn is replacement of lost skin with autografts and restoration of function. In extensively burned patients, the use of homografts[4] provides a temporary respite from continuing infection and nutritional losses and may often be life-saving. Eventually, however, autografting must be accomplished; and when multi-staged grafting is necessary, it is essential that it proceed as rapidly as possible. There is an optimum period from the end of the third through the sixth week when granulations will accept grafts in spite of negative nitrogen balance, local infection, and some nutritional depletion. After this time, fibrosis of granulations, more resistant infections, and further nutritional depletion make subsequent grafting much less successful and greatly prolong the hazards of complications and the period of hospitalization.

DISCUSSION

Care of thermal burns remains one of the most difficult problems in surgery. It has become apparent that severely burned patients can be carried through the initial phase or period of shock, even with extensive third degree injury. This has been made possible by newer knowledge and use of plasma expanders, electrolytes, and blood replacement. Many workers are responsible for this accomplishment and have systematized the initial parenteral and systemic therapy to such an extent that in many instances it now becomes a somewhat routine procedure. The exceptions, however, are elderly patients, children, and those individuals who have secondary associated problems. The patient who has a cerebro-vascular accident, falls into the fire and receives a so-called minor, superficial, 10 to 20 per cent burn is an example of the latter. The patient might survive either alone, but the combined effect may be sufficient to keep the patient from surviving.

Although a patient may survive the initial "shock" phase, there are yet many obstacles in the path to recovery. The time-consuming removal of destroyed tissues, the difficulties in maintaining nutritional balance, and the avoidance of disastrous complications in the interval before autografting remain incompletely solved problems. (Teams of physicians especially interested in burns have done much to minimize these problems, and the establishment of centers for burn treatment should be encouraged.) The manpower demands on both professional and semi-professional personnel are enormous in the treatment of a single serious burn, but properly planned facilities used in the treatment of a large number of patients can reduce these demands.

It is believed that, in the event of mass casualties, present medical facilities would not be equal to the task. Attempts have been made to distinguish the less seriously injured from the hopelessly burned, but the large group in between these extremes will present problems in classification.

The prolonged period of hospitalization and the great expense of treatment of such patients are problems that are incompletely handled by existing agencies. Medical disaster insurance plans are becoming more widely available, and it is hoped that this will be a partial solution to this problem.

Resurfacing of the denuded area of the body and restoration of function is the ultimate aim in treatment. This is most severely handicapped by the uncontrolled infection and the shortage of available skin in those individuals with extensive skin loss. Improvements must be continued in our handling of these problems, if future progress is to be expected in the management of burns.

The most urgent need, however, is public education in safety measures to reduce the incidence of these catastrophic and largely preventable accidents.

SUMMARY

1. The problem of the treatment of serious burns has been stressed, with emphasis on the initial treatment.

2. It is essential that a thorough knowledge of the disturbed physiologic principles be understood before undertaking the treatment of extensive burns.

3. The mortality and morbidity have not been lessened except in that group of patients who are considered borderline and who would usually be treated indifferently.

4. The major problems in the recovery period are those directly related to complications of the burn and the associated diseases unrelated to the burn.

5. Infections of much magnitude continue to be seen and they account for a large number of the deaths occurring in the intermediate and late periods. Unfortunately, antibiotics have not eliminated this severe complication and it is questionable as to whether anything has been accomplished except in the occasional patient.

6. Maintenance of nutritional balance which must be begun immediately, cannot always be accomplished satisfactorily. The mechanism of accomplishing this goal is not important—provided it can be done.

7. Early replacement of the lost skin with a graft is the ultimate goal. The lack of suitable donor site is a deterrent to this.

8. Dermal substitutes have, as yet, been entirely unsatisfactory.

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MIXED TUMOR OF SALIVARY TISSUE—A HARMFUL MISNOMER FOR A CARCINOMA*

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Tumors of the salivary glands have puzzled pathologists ever since they were first described over 100 years ago in the early days of microscopic pathology. The tumors that are now usually called "mixed" have caused the greatest difficulty. The frank adenocarcinomas and epidermoid carcinomas, each with minimal amounts of stroma and each with important potentiality for lymph node and distant metastases, have caused little difficulty in the last 50 years. There have been and still are two important points of disagreement in regard to "mixed" tumors. The first is the interpretation of the varied patterns in the stroma and the second the interpretation of the presence or absence of malignancy.

THE STROMA OF "MIXED" TUMORS

The stroma of these tumors has many patterns. Large quantities of mucinous or cartilaginous material or both are characteristically present. Förster[1] in 1858 and Billroth[2] in 1859 described the mucoid and cartilaginous stroma of these tumors and named then enchondromas. Following their lead in the nineteenth century the stroma was considered by many to be the the most important part of the tumor. The first mention of the name "mixed tumor" was by Minssen in 1874[3] although Förster had come close to this term with "mixed enchondroma." Following C. Kaufman[4] in 1881 these tumors were throught to be myxomatous or chondromatous but, later, an endothelial origin was widely accepted. In addition, many authors believed that the epithelial elements were derived by metaplasia from connective tissue or endothelial cells and not from the glandular cells. Others maintained that both the epithelial cells and the connective tissue or endothelial cells took part in the neoplastic process.

The first suggestion that this peculiar stroma might derive as a result of secretions from the epithelial cells was clearly presented by Ehrich[5] in 1906. He postulated that both the mucinous and cartilaginous material in the stroma were derived from secretions of the epithelial cells. In 1921 Böttner,[6] a student of Ehrich's, came out frankly in favor of this concept in an article entitled "The Secreting Epithelioma (the so-called mixed tumors) of the Oral Salivary Glands." He was the earliest author to call the name "mixed tumor" a misnomer because the stroma in itself had, in his opinion, no primary role in the neoplastic process. This clear expression has not been even discussed in many important, later articles.

Three very important pathologists, however, accepted this idea as the explanation of the peculiar histology. First, E. Kaufman,[7] in his pathology, accepted Böttner's ideas just one year after Böttner's original paper. Secondly, Ewing[8] in the third edition of his book "Neoplastic Diseases" in 1929 refers to Ehrich's work and states: "The mucoid material is a secretion from the epithelial cells, and its diffusion prepares the way for the dissemination of modified glandular and pavement epithelium. Squamous metaplasia occurs readily in the proliferating cells of both ducts and acini, as in pure carcimomas." Thirdly, Willis,[9] in his "Pathology of Tumors," objects to the term "mixed" tumors and suggests that they be called pleomorphic tumors. He stresses the importance of the epithelial secretion. He also points out that the variable tissue response (mucinous or cartilaginous) in the stroma may be on one hand related to the proportions of the two chief substances secreted by the epithelial cells, mucin and ptyalin. At the same time, as was pointed out earlier by Fry,[10] the difference in the histological appearance may also, in part, be determined by nutri-

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Presented during the Baltimore Assembly of The Southeastern Surgical Congress, March 10-13, 1958, Baltimore, Maryland.

tional factors, since there is a deficiency of blood vessels in many large mucinous areas. Ackerman and del Regato[11] state in referring to mixed salivary tumors, "Epithelium in the ducts may be mucin secreting but more frequently there is mucoid degeneration of the stroma."

Among the pathologists who do not discuss the significance of the secretion of mucus from the epithelial cells of "mixed tumors" are Foote and Frazell.[12] However, they do indicate that "mixed" tumors frequently contain a mucinous substance—(on page 104 in describing the gross appearance of "adenoid cystic carcinomas" they state: "Varying degrees of hyaline trabeculations are seen from time to time; and whereas the cut surfaces are usually moist, they seldom produce the *slimy or chondroid aspect common to many benign "mixed tumors"*). They also stress the importance of epithelial mucoid secretions in the "mucoepidermoid tumors" and mucoepidermod carcinomas. This group they have split off and described as an entity separate from the "mixed" tumors.

Some of the modern standard text books of pathology ignore the possibility that epithelial secretions may be the cause of the stromal pattern.[13, 14, 15] However, Anderson,[16] Smith and Gault[17] and Ackerman[18] do recognize that either the epithelial cells or the stroma produce secretions. Anderson discusses the possible effect of secreted mucus on the stroma.

Mulligan[19] separates from the group of mixed tumors a group of cases he calls mucoepidermoid adenoma in which he recognized the rupture of ducts and the discharge of material into the stroma. These are the same type of cases called mucoepidermoid carcinoma by Foote and Frazell. Ward and Hendrick,[20] Ackerman and del Regato[11] and Cutler and Buschke[21] in their books on the diagnosis and treatment of cancer and Garcelon,[22] in his chapter on salivary tumors in the book, "Cancer, A Manual for Practitioners" also pay no attention to a possible secretory function of these tumors. Among the recent text books on surgery, those of Cole,[23] Christopher,[24] Richards,[25] Thorek[26] and Ferguson[27] have nothing on secretion. However, Blair and Olch[28] do mention the secretory theory as a possibility referring to Krompecker[29] as making this suggestion. Also Berman[30] quotes Ehrich[5] as stating that parotid epithelial cells undergo mucoid degeneration in these tumors.

Evidence that the stroma of these tumors is reactive may be summarized as follows:

1. Sarcoms practically never occur.
2. When recurrence or metastasis occurs as a rapidly growing carcinoma the peculiar stroma is no longer present.
3. "Mixed tumors" also occur in other organs whose epithelial cells have secretory functions. Examples are "mixed tumors" in the oropharynx, the lachrymal glands, skin adnexal glands, bronchial glands and the breast.

On the other hand histochemical evidence is not yet conclusive on the point of escape of epithelial secretions into the stroma.

ARE MIXED TUMORS MALIGNANT?

Almost all authors, pathologists and surgeons alike, divide "mixed" tumors of salivary glands into two classes, benign and malignant, with the first class comprising the great majority of the cases. However, Willis,[9] Trueblood[31] and Krompecker[29] and a very few others consider all "mixed" salivary tumors to be malignant. The latter author called them basal cell carcinomas. A few authors hedge. Thus Mettler,[32] Berman[30] and Clarke[33] have considered them to be either potentially malignant or intermediate in status between malignancy and benignity. One author discussing this point stated "they were among the most malignant of the benign tumors" and that salivary carcinomas were "among the most benign of the carcinomas." Although McFarland divides them into benign and malignant groups he admits that pathologists really cannot tell which group to place any one case in. He had 25 "competent" pathologists study the slides on 25 cases and found that their decisions as to the malignancy of the individual mixed tumors might as well have been made by throwing dice. (He did not say this but that is what his tabulation indicates.)

The pathological diagnosis of malignancy in these tumors is fraught with difficulties. The obviously malignant "mixed tumors" exhibit marked anaplasia of cells, increased and abnormal mitotic figures, invasion of capsule and surrounding tissue and perineal and vascular permeation. These tumors present no difficulties. However, for the great majority of "mixed tumors" it is recognized that a significant *hiatus* exists between their biological behavior and their relatively benign morphological appear-

ance. In addition, the obvious difficulty of locating the most malignant foci in any specimen makes it obvious that one cannot rely on morphologic interpretation as a guide to benignity or malignancy. These considerations should lead the surgeon to consider all such tumors as either malignant or as carcinoma *in situ* if he is to obtain the best therapeutic results.

Willis[9] states as follows: "Because of their supposed encapsulation and the rarity of metastasis, the pleomorphic salivary tumors have often been classed as "benign." This fallacious idea cannot be too strongly discouraged. Tumors which are often demonstrably not encapsulated, which frequently recur after removal, which may widely infiltrate surrounding tissues, and which occasionally metastasize are far from "benign."

Figures 37 to 43 in Foote and Frazell's fascicle[11] illustrate beautifully the microscopic pictures of instances of breaks in capsules and infiltration into tissues of tumors they call benign. Figures 44 and 45 illustrate the structure of a tumor they call "histologically benign" with its lung metastasis! On the other hand, mitotic figures are very rare in the cases called "benign." This surely is related to the usual very slow rate of growth. The degree of differentiation is very great, even though the microscopic appearance is very variable from case to case and from one place to another in the same specimen. It is these two items that have been the strongest ones in influencing pathologists to consider these cases benign.

Recurrence after removal varies with the state of the tumor and the ability of the surgeon in this field of endeavor. Almost all recent articles in periodicals advise a carefully performed lobectomy or parotidectomy with preservation of the nerve in parotid cases and complete removal of the submaxillary gland in cases arising in that gland. Recent reports of results after such operations on primary "benign mixed tumors" show recurrence rates of 2 to 5 per cent. After such operations on patients already recurrent, chance of further recurrence increases to 15 to 25 per cent.

The recurrence rate after lesser operations is not known but it is believed to be high because 25 to 40 per cent of the patients that come to the larger clinics for the first time have had previous operations. This situation is totally different from that of clearly benign tumors. In spite of the fact that fibroadenomas of the breast and lipomas have no capsule, "shelling" them out cures practically all cases. In the occasional arborescent lipoma a small projecting lobule may be left behind, but this does no real harm as a second operation will almost certainly cure it.

This brings us to consider the sixth item, subsequent behavior of the tumor. When "benign" salivary tumors are neglected, carcinoma may arise in them. This is also illustrated by Foote and Frazell[11] in figures 52 to 63. Many other authors have commented on the fact that when "benign" lesions recur they are likely to recur as malignant "mixed" tumors or as frank carcinoma. If serial recurrences occur the lesion is likely to become more and more active as time goes on.

The decision as to whether all these tumors should be called malignant or not really lies in the field of semantics. It is not a serious decision from the standpoint of the surgeon who had studied the subject thoroughly and has a full understanding of the pathology and biology of these tumors. However, it is disastrous to name these tumors in such a way that the name minimizes their seriousness and encourages the less knowledgable surgeon or general practitioner to do such a limited operation that the one best chance for cure with preservation of the nerve is irrevocably lost.

If the name given to a tumor connotes that it is non-malignant, the tendency for the doctor who writes about it is to suggest a minimum amount of surgery. If he does this and an inexperienced surgeon follows his advice he will perhaps do even less than the writer suggests. At present the most modern articles in surgical journals advise a lobectomy as the minimum operation for a small, superficial parotid tumor and a total removal of the gland for larger or deep tumors and for all submaxillary tumors. But the authors of various modern books on surgery are quite different from the authors of articles in the surgical journals.[34, 35, 36, 37, 38, 39] This is important because the surgeons who need the best advice are not as likely to look up articles scattered in various journals as to turn to a book. What is found in the latest books? (Older ones are worse.)

Garcelon,[22] Blair and Olch[28] and Berman[30] recommend the above treatment. However, on the other hand, Ward and Hendrich,[19] Ackerman and Del Regato[11] Cutler and Buske,[21] Cole,[23] Christopher,[24] and Richards[25] advise enucleating these tumors when they are small ones. Richards

in *Surgery for General Practice* and Ferguson[27] in *Surgery for the Ambulatory Patient*, advise enucleating these tumors under local anaesthesia in an ambulatory patient.

Finally, Thorek[26] in *Surgical Errors and Safeguards* advises enucleating them *within their capsules*. No wonder Baehrs[40] reported to this Society last year that 40 per cent of the new cases coming to the Mayo Clinic came for the treatment of a recurrence following surgery performed elsewhere.

WHAT SHOULD THE VARIOUS SALIVARY TUMORS BE CALLED?

In the first place it is essential that the word "mixed" be dropped once and for all despite its universal acceptance for 80 years. As indicated above this part of the name is a holdover from the days when all pathologists were confused by the irregular stroma and considered that these tumors developed as a result of a neoplastic process in the stroma simultaneous with the neoplastic changes in the epithelial cells. It has absolutely no meaning if the stromal changes are believed to be a reaction to secretion from the epithelial cells.

If we are to indicate the malignancy of these tumors in their names, we must in some way separate them from the more actively growing group already called carcinoma. The over-subdivided classification recommended by many pathologists seems to serve little usefulness when the prognosis of the patients put in the subclasses is similar. The following classification separates these tumors on the development of glandlike or epidermoid structures. It also provides a reasonable separation of the more differentiated from the more anaplastic tumors.

SUGGESTED CLASSIFICATION OF SALIVARY CARCINOMAS

1. Adenocarcinoma *in situ*
2. Secreting epidermoid carcinoma
3. Adenocarcinoma
4. Epidermoid carcinoma
5. Carcinoma simplex

Class 1 inclubes all the cases classified by Foote and Frazelle[11] as benign mixed tumors, malignant mixed tumors, adenoid cystic carcinoma, acinic cell adenocarcinoma, mucous cell adenocarcinoma, papillary cystadenoma lymphomatosum, oncocytoma, benign lymphoepithelial lesion and adenocarcinoma with pseudoadamantine pattern. It would correspond also to the cases classified by Willis,[9] as pleomorphic adenoma and pleomorphic adenocarcinoma. Class 2 is the same group called mucoepidermoid carcinoma by Foote and Frazell. The 3 and 4 groups are self explanatory. The cells are nonsecretory and, therefore, the stromas are similar to the stromas of adenocarcinoma or epidermoid carcinomas arising in other organs. Because the cells are so primitive they do not secrete, they are much more actively malignant and have a greater tendency to regional and distant metastasis. Foote and Frazell subdivide the adenocarcinomas into trabecular or solid adenocarcinoma and anaplastic adenocarcinoma. They call the cases we have named epidermoid carcinoma by the name squamous cell carcinoma, which names they and other pathologists consider to be identical in present day usage.

Class 5 is made up of the most anaplastic carcinomas with no differentiation. Some of these look very much like anaplastic sarcomas and have been so named by many authors. It is possible that true fibrosarcomas may very rarely arise in the stroma of parotid glands but the occurrence is so rare that Foote and Frazell[11] do not discuss or illustrate them. However, in their tabulation of the 877 cases of salivary tumors studied at the Memorial Hospital from 1930 to 1949 they record 37 cases as malignant, unclassified tumors. Perhaps these include some sarcomas but they do not illustrate or discuss these cases either. The authors prefer to consider such cases, until some grounds for a better, more accurate diagnosis arise, as carcinoma simplex.

In classifying all the tumors of the salivary glands, a difficulty arises because parts of the tumor may be quite different from each other. In case areas of classes 3, 4 or 5 are seen side by side with classes 1 or 2, the tumor should be labeled according to the most undifferentiated part and should be treated accordingly.

TREATMENT

It is not the province of this paper to go into the details of treatment. Excellent detailed descriptions of the anatomy may be found in the article by Davis *et al.*[41] and of the operative technique in the article by Brintnall *et al.*[42]

In no case should a biopsy be made for fear of spreading malignant cells into the tissues and of missing the most active part of the lesion. In all cases the operation should be done under full gen-

eral anesthesia and the nerve exposed at the start of the operation. Not less than a superficial lobectomy of the parotid should be done for the more accessible tumors of the parotid. For deep tumors a total parotidectomy is advised. For submaxillary tumors a total removal of the gland is advised. In performing any of these operations the dissection must be carried out through clearly normal gland or adjacent tissue in all directions and the tumor must never be grasped with tenaculums, toothed forceps, Kocher clamps or any other biting instrument. In other words, they must be handled with all the care that is indicated for any other malignant tumor.

The above described local operation is all that is indicated for pathological classes 1 and 2 above, but classes 3, 4 and 5 should always have total removal of the involved gland, any adjacent tissues that are suspected of involvement and a complete unilateral neck dissection if the best chance of cure is to be achieved and if an attempt at cure is advisable.

In Sweden many authors[43] and in England some authors recommend x-ray treatment as an adjunct to minor surgery. This item is mentioned only to condemn it. These slowly growing tumors are most insensitive to radiation and the evidence presented is not convincing.

SUMMARY

1. Evidence has been presented that "mixed tumors" of the parotid may have their peculiar stroma as a result of secretions from the epithelial cells permeating the stroma.

2. None of these tumors should be called benign, but most authors do so name them.

3. Many text books of surgery not only call most of these tumors benign but advise minor surgical treatment.

4. It is believed that the name and this advice together are responsible for most of the many patients that come to the specialized clinics with established recurrences.

Acknowledgment. The authors express appreciation to Dr. Oscar Hirsch for aid in this study.

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VOLVULUS OF THE CECUM COMPLICATING PREGNANCY: A REPORT OF THREE CASES*

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Volvulus of the cecum is an infrequent cause of intestinal obstruction, only a few hundred cases having appeared in the world literature. As a rule, these articles have consisted of a brief review with addition of the author's personal experience with one or more cases. Several case histories of cecal volvulus complicating pregnancy have been reported, each emphasizing the diagnostic difficulties encountered which often have delayed surgical treatment.[1, 2, 3, 4, 5] Three cases of volvulus occurring during labor or the early postpartum period are presented in this report.

Hypermobility of the right colon is generally accepted as the predisposing cause of volvulus. Such a situation is the result of incomplete rotation, or inadequate fixation of the cecum and ascending colon to the posterior abdominal wall. Since many people are shown by autopsy, or radiologic studies, to have sufficient mobility of the right colon to permit twisting, there must be other inciting or precipitating mechanisms which actually set the process in motion. Postoperative adhesions, constipation, hyperperistalsis, trauma and pregnancy may be significant. The torsion often involves the terminal ielum and ascending colon, inasmuch as these structures share a common mesentery. The axis of rotation may be transverse, oblique or parallel to the cecum, and may be clockwise or counter-clockwise. This produces a closed loop obstruction with rapidly rising intraluminal pressure and massive distention of the segment. Obviously, ischemia and perforation of the cecal wall is invariable unless early surgical treatment is employed. Volvulus of the entire right colon and some part of the small intestine was found in each of the three cases here reported. Tearing of the serosa of the cecum with impending perforation was found in two instances, and in the third, necrosis of the entire right colon, ileum and majority of the jejunum was encountered.

* Presented during the Baltimore Assembly of The Southeastern Surgical Congress, March 10-13, 1958, Baltimore, Maryland.

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The signs and symptoms of cecal volvulus are those of intestinal obstruction and vary with the duration of the process and state of the bowel wall. Cramping pain is noted initially and may be poorly localized; reflex vomiting is usually seen early. Pain becomes constant and peritoneal irritation prominent as ischemia of the bowel wall increases. Fever, leukocytosis and tachycardia often herald impending necrosis of the involved segments. The distended bowel often may be palpated, particularly in the flaccid, postpartum abdomen.

An accurate diagnosis of cecal volvulus is seldom made on the basis of history and physical findings alone. The situation is even more dangerous when it occurs during pregnancy, labor, or during the immediate postpartum period. In such instances, the gravid uterus may interfere with abdominal examination; the pains of labor, or analgesic medications may further confuse the examiner. These facts are attested by dangerous delays in the recognition and treatment of volvulus.

Roentgenography may be the most important factor in the detection of volvulus of the cecum. Usually, plain films supine and erect, without the use of contrast media are entirely satisfactory. The utilization of barium is advisable only when serious difficulty in differentiation occurs. The presence of a markedly distended cecum without corresponding distention of the remaining colon should suggest the diagnosis of mechanical obstruction. The distended cecum often lies in the left upper quadrant and a fluid level may be seen in the erect position (figs. 1 & 2). A dilated cecum occupying the left upper abdomen may actually displace the stomach and differentiation may be difficult. If a barium enema is utilized, a cone shaped obstruction with spiral mucosal folds at the level of the ascending or right transverse colon is diagnostic. The barium will stop at the point of torsion, at which site the typical mucosal pattern indicative of twisting may be visualized.

Treatment requires prompt surgical correction of the twisted bowel. Simple detorsion suffices if

FIG. 1. Markedly distended bowel, representing cecum and adjacent ascending colon and hepatic flexure, unusually placed in the left upper adbominal quadrant, with fluid levels in the erect position. This is the classical appearance of obstruction with volvulus of the right colon, so often associated with a mobile cecum or incomplete rotation.

FIG. 2. Cecal distention with a fluid level seen in the erect position. Early in cecal volvulus, a single fluid level is frequently noted. Obstruction of sufficient duration has resulted in gas distention and fluid filled adjacent small bowel loops. Note the absence of distention of the distal colon. Usually, only such plain films are required for diagnosis. Barium contrast studies may be utilized if serious difficulty in differentiation occurs.

the process is seen before the bowel wall is compromised by over distention and infarction. Usually the cecum may be untwisted and fixed by sutures, with or without cecostomy in an appropriate abdominal quadrant. A tightly distended, ischemic cecum may require decompression before it can be safely untwisted. A needle attached to suction may successfully accomplish decompression and allow the bowel to be handled with greater safety. The question of routine cecostomy is unanswered but the simplicity of its performance makes it desirable, particularly in those instances where serosal tears resulting from overdistention are found. Obviously, resection is necessary for those cases having advanced to gangrene. If the patient's condition permits, resection with primary ileocolostomy would be the procedure of choice, but situations may occur when exteriorization of the gangrenous bowel will be life saving.

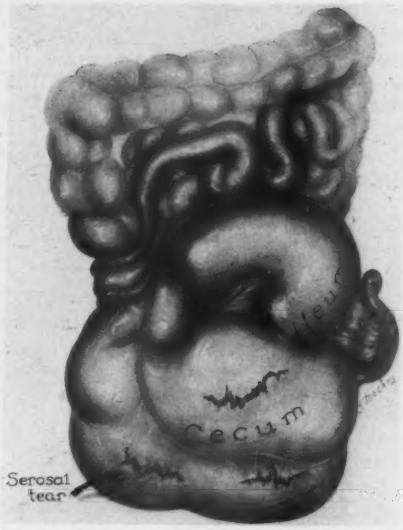

FIG. 3. (Case 1.) Volvulus of cecum and right colon with marked distention of cecum and terminal ileum.

CASE REPORTS

Case 1. Dr. H. S., a 24-year-old gravida two, para one, was delivered of a healthy male infant after an 8-hr. uneventful labor. She was comfortable until 18 hr. postpartum, at which time she experienced a gradual onset of intermittent pain in the right lumbar region. Within an hour the pain had become excruciating, and was only partially relieved by narcotics. Examination revealed moderate generalized distention with hypoactive peristalsis. Nausea was present but there was no vomiting. Intravenous urography revealed no abnormality. Supine films of the abdomen demonstrated distention of the right colon without dilation of the small bowel. Throughout the next few hours abdominal pain increased in intensity and was more prominent in the right lumbar area and radiated to the back. Twelve hours after the onset of pain a barium enema revealed a block at the hepatic flexure. A greatly distended cecum and ascending colon were identified and a presumptive diagnosis of volvulus of the right colon was made. Examination just prior to surgery revealed a pulse rate of 76, temperature of 99°, and a white blood count of 17,700. The abdomen was soft and a large tender mass was found in the epigastrium. The mass was movable and was tympanitic to percussion. Laparotomy was performed 15 hr. after the onset of pain and revealed a 360° volvulus involving the entire ascending colon, cecum and 12 in. of the terminal ileum, (fig. 3). The clockwise volvulus was untwisted and the cecum fixed in the right lower quadrant by tube cecostomy. The postoperative course was uneventful and the patient was dismissed on the 15th day following surgery. The cecostomy, however, did not close spontaneously because of a sharp kink located at the hepatic flexure of the colon at the exact site of the previous torsion. Six months after dismissal from the hospital a right colectomy was performed. Bowel continuity was restored by end-to-end ileo-transverse colostomy. Following this operation, the patient has remained well for 3 years.

Case 2. Mrs. J. C., a 35-year-old gravida two, para one, was delivered of a normal male infant after an uneventful labor of 8 hr. duration. Her only complaint during the period of gestation was occasional mild abdominal distention and cramping pain which was attributed to "gas." The past history is significant in that a diagnosis of appendicitis was made at the age of 16, at which time the abdomen was entered through a McBurney incision and the appendix was never found. During the morning of the first postpartum day she complained of headache, lower abdominal cramps and mild distention. There was a desire to expel flatus which was aggravated by walking. Following an enema, flatus was expelled and the patient experienced some relief. On the second postpartum day abdominal distention was again noted, although the discomfort was minimal. During the third day abdominal distention became progressively worse, and there was recurrence of constant lower abdominal pain associated with nausea and vomiting. On the morning of the fourth postpartum day, a

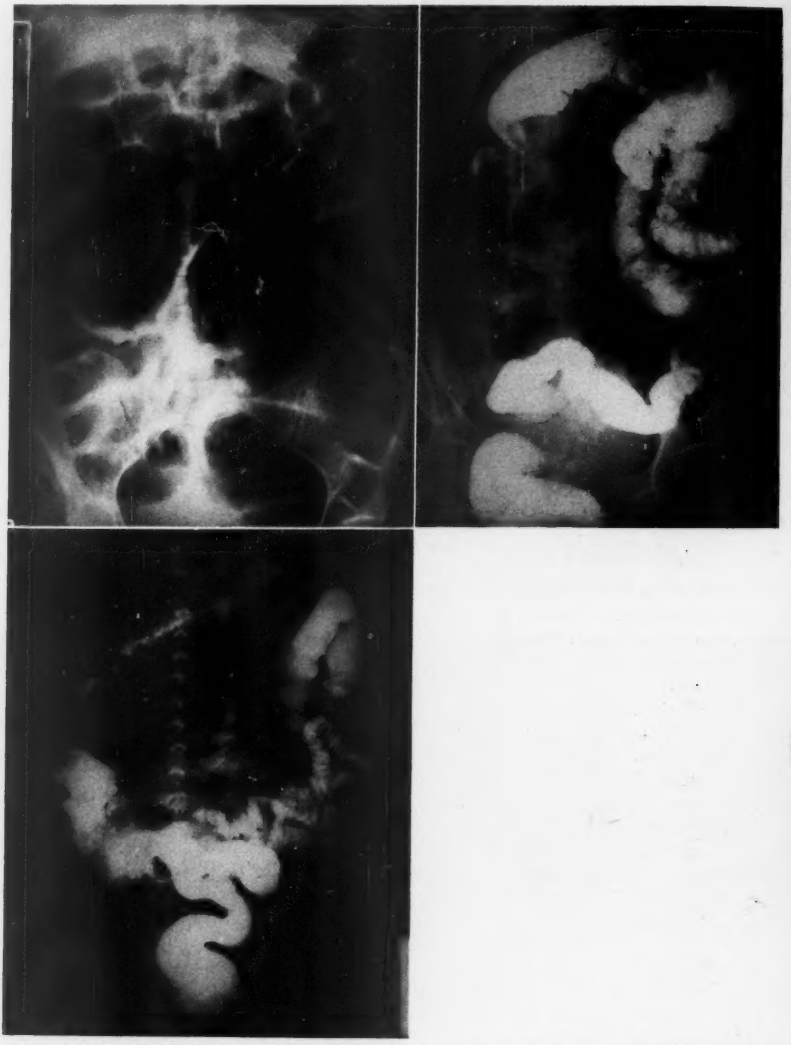

FIG. 4 (*Top left*). Flat plate reveals marked distention of colonic loops within the left abdomen. The distal colon seems relatively free of gas. Distended small bowel loops are also present. Findings consistent with volvulus of the right colon in a patient with a mobile cecum or incomplete rotation. (*Top right*). Barium enema clearly demonstrates obstruction with the colon at the hepatic flexure, with a spiral configuration diagnostic of volvulus. (*Bottom*). Post-operative barium study reveals the cecum in usual position in the right lower abdominal quadrant following surgical fixation. Note the cecostomy tube.

roentgenogram demonstrated a markedly dilated cecum lying in the left upper quadrant and a barium enema revealed a block in the hepatic flexure typical of torsion (fig. 4 *a*, *b* & *c*). At this time the pulse rate was 108, blood pressure 130/80, and leukocyte count 11,100. The abdomen was distended and a tender spherical mass occupied the entire epigastrium. Exploratory laparotomy was carried out and cecal volvulus was found. The cecum was tightly distended and there were several long serosal tears present on its surface. Aspiration was necessary before any attempt at untwisting could be carried out. The ascending colon, cecum and terminal ileum were found to

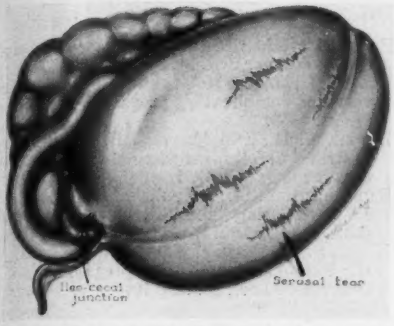

FIG. 5. (Case 2.) Massive distention resulting from volvulus of cecum and right colon.

FIG. 6. (Case 3.) Volvulus of practically entire midgut with infarction and gangrene—requiring extensive resection.

have undergone clockwise rotation of 360° at a point just distal to the hepatic flexure (fig. 5). Although there was ischemia of both large and small bowel, resection was unnecessary. Following reduction of the torsion, a cecostomy was performed and the catheter was brought out through a stab incision in the right lower quadrant, the cecum having been sutured to the parietal peritoneum in that area. The postoperative course was uneventful, cecostomy closed spontaneously, and the patient has been well for some 18 months following surgery.

Case 3. Mrs. W. H., a 21-year-old gravida three, para two, was admitted with a diagnosis of early term labor. Lower abdominal pain had been present for 4 hr. and was increasing in severity. The pain was constant but was intensified by cramps occurring at intervals of 5 to 10 min., which were thought to be the pains of labor. Mild nausea was present but no vomiting had occurred. The pulse rate was 88 and the blood pressure was 140/90. The abdomen was distended and peristalsis was present although hypoactive. Uterine contractions were of poor quality but appeared to be associated with an increase in abdominal pain. The fetal heart rate was 160 per min. A rectal examination was performed and the cervix could not be palpated satisfactorily; a sterile vaginal examination revealed a non-dilated cervix. At the end of four hours there was no appreciable change noted in the patient's condition. Five hours after admission, pulse rate rose abruptly to 160, the blood pressure suddenly became unobtainable, and the fetal heart tones inaudible. The abdomen was now quiet and tenderness was elicited throughout. The uterus was palpated with some difficulty and there was a question of free fluid within the abdominal cavity. A presumptive diagnosis of ruptured uterus was considered and preparations made for immediate surgery, which was accomplished exactly 6 hr. after admission. The abdominal cavity was entered through a low midline incision, a classical Cesarean section was performed, and a dead fetus was removed. Following completion of Cesarean section the abdominal viscera were examined and the lower jejunum, entire ileum, cecum, ascending and right transverse colon were found to have undergone a clockwise volvulus of 720° around the superior mesenteric artery (fig. 6). The bowel involved in the rotation was gangrenous and resection was necessary. Intestinal continuity was restored by end-to-end anastomosis between the jejunum and the transverse colon. As anticipated, the postoperative course was complicated by diarrhea and electrolyte problems. Recovery was prolonged but the patient left the hospital in 17 days and is living an essentially normal life three and one-half years after surgery.

SUMMARY

1. Volvulus of the cecum is a rare cause of intestinal obstruction the underlying basis of which is hypermobility of the cecum and the right colon.

2. Pregnancy and labor may be initiating mechanisms in producing the actual twist.

3. Volvulus occurring during pregnancy or labor may escape early notice and progress unrecognized to perforation or gangrene.

4. Supine roentgenograms of the abdomen will often establish an early correct diagnosis which

demands immediate surgical intervention. Simple untwisting often suffices but resection or exteriorization is required if perforation or gangrene have occurred.

5. Three cases of volvulus of the right colon occurring during labor and early postpartum period are presented.

Acknowledgment. The authors express gratitude to Drs. Lon Grove and William R. King (cases 1 and 3), and Dr. J. Frank Walker, Department of Radiology.

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INTESTINAL OBSTRUCTION SECONDARY TO COCONUT FIBERS*

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"Coconut ileus" is a rare etiologic factor as a cause of intestinal obstruction. Although coconut pulp is well known as a constituent of phytobezoars, in only five instances in the literature has this substance been listed as the etiologic agent in obstruction of the small bowel.[32, 36, 39, 49] For this reason, and because a moderate sized Meckel's diverticulum was present and involved in the obstructive process, the following case is reported.

CASE REPORT

The present case is that of a 30-year-old male electrician with the chief complaint of colicky abdominal pain referred to the vicinity of, and inferior to, the umbilicus. The pain was of 2 days' duration and was suddenly accentuated the day of hospital admission. Nausea and vomiting were present for 12 hours immediately prior to admission. The vomitus was yellow in color and at no time contained any blood or "coffee-ground" material. There had been no bowel movements or passage of flatus during this period.

Upon inquiry as to the foods consumed in the past 24 hours, no mention of desserts was made. Questioning revealed that the patient had been under treatment 3 years for duodenal ulcers, confirmed on three previous occasions by x-ray.

Laboratory data. Examination of the blood revealed the hemoglobin to be 16.5 gm., the red blood cell count 5.28 million, and the white blood cell count 10.4 thousand. The differential white blood cell count revealed the following: 75 per cent segmental, 7 per cent bands, 13 per cent lymphocytes, 4 per cent monocytes and 1 per cent eosinophils. The urine was clear, straw-colored, and with a specific gravity of 1.018. Protein, sugar and microscopic examination were all negative.

X-ray examination of the chest and abdomen showed the diaphragm, heart, and lungs to be normal. The upright and recumbent films of the abdomen (fig. 1) showed gas-distended bowel in the left midabdomen and in both upper quadrants. The upright position showed definite fluid levels, suggesting mechanical small bowel obstruction in the distal ileum. The patient was taken to surgery approximately 12 hours after the onset of his present illness, with a preoperative diagnosis of mechanical small bowel obstruction. Exploration was carried out through a right lower rectus incision. Upon entering the peritoneal cavity straw-colored fluid was encountered and 500 cc. were removed. The cecum was identified and the appendix was brought into view. It presented no gross evidence of pathologic change. The ileum was identified at the ileocecal junction and was followed back 8 inches, where a rather soft intraluminal obstructing mass was encountered. This was considered to be a food bolus and measured 4 cm. in diameter. Proximally, the intestine was moderately distended, and at about 16 inches from the ileocecal valve a distended Meckel's diverticulum was found. The base of the diverticulum measured about 3.5 cm. and its length was about 4 cm. The diverticulus was located on the antimesenteric border of the ileum and was supplied with its own mesentery (fig. 2). Inspection of the remainder of the small bowel to the ligament of Treitz revealed no other abnormalities. The duodenum and stomach appeared unremarkable.

Palpation of the Meckel's diverticulum suggested that its contents were similar to that causing the ileal obstruction. The obstructing bolus was then milked proximally into the Meckel's diverticulum, about 6 inches of ileum containing the diverticulum was resected, and an end-to-end anastomosis was performed. Prior to the anastomosis large amounts of coconut strands were removed, both proximally and distally. The cecum was then delivered and an appendectomy was performed with inversion of the stump.

Surgical pathology. The specimen consisted of a resected portion of ileum, including a Meckel's diverticulum. The ileal segment measured 8 cm. in length, and the diverticulum measured 6 by 4 by 2.5 cm. The serosal surface of the specimen was gray-pink and glistening, with patchy, lusterless areas. When the specimen along the antimesenteric border was opened the diverticulum and the distal portion of the ileal segment appeared to be tightly packed with shredded, white, fiber-like material resembling coconut (fig. 3). Microscopic sections of the diverticulum revealed compressed mucosa of ileal type. The deeper portions of the wall were not remarkable. Sections of the ileum presented an essentially normal pattern. A discoid portion of soft, gray-pink tissue from the region of the di-

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FIG. 1. X-ray films revealing evidence of mechanical small bowel obstruction.

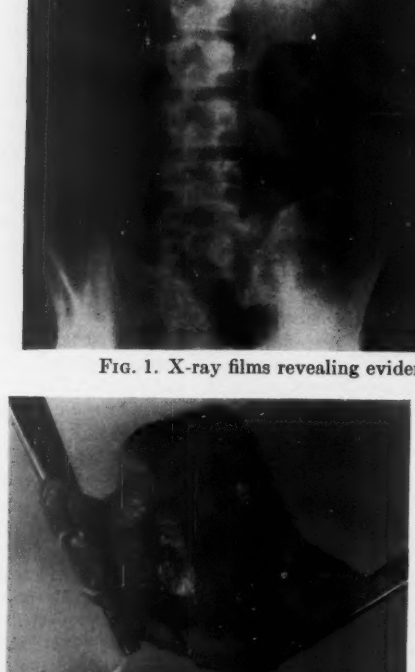

FIG. 2. Meckel's diverticulum. Removed strands of coconut fiber can be seen in the lumen.

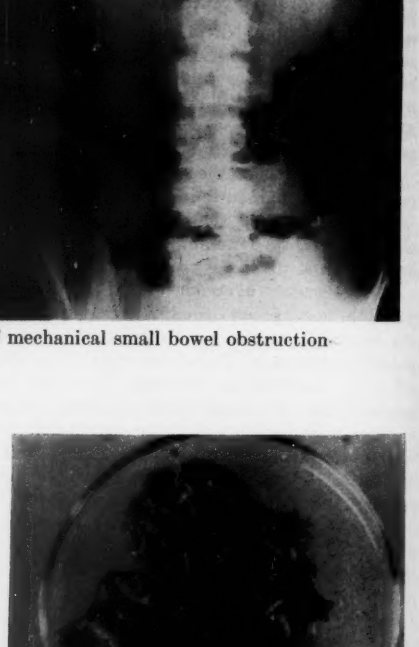

FIG. 3. Meckel's diverticulum, opened, revealing the coconut bolus.

TABLE 1

Food substances reported in literature as etiologic agents in the production of intestinal obstruction

Material	No. of Cases
Persimmons (not true food)	30
Oranges	17
Peaches	18
Mushrooms	14
Figs	10
Cherries (some including stones)	10
Beans	9
Bran	8
Unspecified fruits	8
Grapes	8
Apples	7
Potatoes	5
Ammunition bread (*Kriegsbret*)	5
Coconut (raw)	3 (raw)
Apricots	3
Carrots	3
Oats	3
Pears	3
Watermelon seeds	3
Gooseberries	3
Poppy seeds	3
Almonds	2
Cucumbers	2
Grasshoppers (*Savteselles*)	2
Peanuts	2
Corn	2
Apples, pears, and peas	2
Poppy seed, beans, and peas	1
Popcorn	1
Raisins	2
Peas, fruit lumps	1
Peas, potato lumps	1
Sauerkraut	1
Banana	1
Bones and peanuts	1
Butterbeans	1
Cantaloupe	1
Chicken skins	1
Fish scales	1
Lettuce leaves	1
Orange and seeds	1
Radish and garlic bean	1
Sweet potato	1
Prunes	1
Turtle eggs	1
Watercress	2
Garlic bulb	1
Celery	1
Collards	1
Monkey nuts	1

verticulum measured 1.5 by 0.8 by 1.3 cm. and revealed, on gross and microscopic examination, the pattern of a lipoma. An appendix, 6 cm. long by 0.6 cm. in diameter, was also included in the specimen submitted to the pathology laboratory and likewise revealed no gross or microscopic changes.

Pathologic diagnosis. Segment of ileum, coconut impaction in a Meckel's diverticulum; mesentery of ileum, lipoma; appendix, histologic.

Subsequent questioning revealed the patient's favorite dessert to be coconut pie. Two days prior to the onset of the symptoms he had eaten a portion of coconut pie and repeated the process 1 day later. In all, he had consumed the greater portion of a pie, containing approximately 8 ounces of refined coconut.

Postoperative course. The postoperative course was uneventful The patient began to pass flatus during the first postoperative day, and on the third and fourth days numerous strands of coconut fiber were present in the feces. He was discharged from the hospital on the seventh postoperative day.

DISCUSSION

Elliot[18] in 1932 reviewed the literature pertaining to bolus obstruction of the intestine, reporting a study of 39 cases since the year 1910. His results indicated conservative management to be the treatment of choice, as operative mortality was 48 per cent. In table 1 is presented a list of the food substances reported in the literature as etiologic agents in the production of intestinal obstruction.

It was observed that the predisposing factors appeared to be (1) increased age with an atonic digestive tract, (2) stricture or neoplasms, (3) the habit of swallowing foods whole, and finally (4) the drinking of a large quantity of water with meal, resulting in swelling of vegetable fibers and consequent increased bulk sufficient to cause obstruction.

The most common point of impaction is about 30 inches proximal to the ileocecal valve, at which point the ileum is at its narrowest.

Symptoms usually occur around 12 hours after ingestion of the obstructing food and are those of acute small bowel obstruction, with characteristically an early and pronounced tympanitis out of proportion to the probable duration of obstruction and apparent good condition of the patient. In late cases the picture is that of progressive

intestinal obstruction with severe vomiting, distention, and a rapid downhill course.

In the course of treatment, if the condition is diagnosed preoperatively, one may try decompression with one of the long lumen tubes and the use of antibiotics, antispasmodics, parenteral fluids, and enemas. If the patient does not make rapid progress operation should not be delayed.

Reports in the literature indicate that the diagnosis is often missed. With the advent of antibiotics, the mortality rate will doubtlessly be decreased in the future for those cases requiring an operation.

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MESENTERIC AND OMENTAL CYSTS*

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Benevieni reputedly first described a mesenteric cyst as an interesting anatomic variant encountered during a post-mortem examination in 1507.[9] In 1897 Moynihan published a classic article reporting nine cases, a few of which had been successfully treated.[9] Recent reports attest to the fact that the condition is not as rare as previously assumed.[4, 6, 8]

A mesenteric or omental cyst is difficult to define and there is no unanimity concerning this point. The most inclusive definition is that of Thompson[10] who states that mesenteric cysts are "cysts which occur in or near the mesentery, which are not malignant, dermoid or parasitic and which do not arise from any normally placed retroperitoneal organ." An omental cyst is indistinguishable histologically and presumably has the same etiology as the mesenteric cysts.

There is some conjecture concerning the etiology of the cysts. Moynihan believed that they resulted from dilation of a portion of a lymphatic vessel occluded as a result of some inflammatory disturbance.[9] Because of the extensive anastomoses of the lymphatic vessels and failure to produce cysts by experimental obstruction of the thoracic duct, this has been disputed.

If the definition of mesenteric cysts proposed by Thompson[10] is adhered to, neoplastic and parasitic cysts are excluded. Dowd has suggested an embryonic origin of the cysts into which there has been an effusion of chyle.[3] The possibility of a traumatic origin in some cases cannot be discounted. Handelsman and Ravitch[6] have postulated the existence of two forms of mesenteric cysts, the chylangioma and the chyle-filled lymphatic cyst which results from obstruction. They regard the chylangioma as a lymphangioma, occurring in embryonic lymphoid centers as do most lymphangiomas. The presence of smooth muscle within the wall of the cyst has been interpreted as indicating a developmental origin. It is possible that mesenteric cysts occur when embryonic lymph sacs persist as such and retain the fluid secreted by the endothelial lining. Because of failure to develop connections with lymphatic channels leading to the cisterna chyli these cysts continue to enlarge until symptoms occur.

From the foregoing it hardly seems logical to ascribe a common mode of origin for all mesenteric or omental cysts.

The classification of mesenteric and omental cysts is similarly difficult. The first classifications were based upon the contents of the cyst and included such terms as chylous, serous, lymphatic and sanguinous. As Warfield has emphasized, the character of the fluid denotes accidents that have befallen the cyst and only occasionally has any bearing on the etiology.[12] Similarly the classification into unilocular and multilocular varieties probably is not justified unless they are distinct histologic entities. Since the etiology is so difficult to ascertain, classification seems unwarranted on this basis.

The gross appearance of a mesenteric cyst at first may resemble that of an enteric duplication but after closer inspection they should be differentiated from each other rather easily. Enteric duplications differ grossly from mesenteric cysts in that they share a common wall and blood supply with the adjacent bowel. Also the wall of a duplication is as thick as the contiguous bowel while mesenteric cyst walls tend to be quite smooth and thin—seldom more than 2 mm. in thickness.[5] The fluid within the cyst may be serous and thin or quite thick and turbid. Occasionally the fluid may be bloody but usually this is caused by preceding trauma.

The histologic appearance of the cysts may be varied but ordinarily they resemble lymphangiomas and consist of endothelial and connective tissue elements. The walls and supporting septa of the multilocular cysts may contain fibrous tissue, round or giant cells, elastic tissue and smooth muscle. The lining usually consists of a flattened layer of endothelial cells which may not be present in all sections. Frequently there are collections of lymphocytes or lymphoid follicles. There is no essential difference in the histologic

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characteristics of the mesenteric and omental cysts.

The cysts may be unilocular or multilocular. Moynihan emphasized the fact that unilocular cysts are more common in the mesocolon whereas the multilocular cysts occur most often in the mesentery proper. The cysts are quite small during the initial stages of development but by the time symptoms occur they usually are quite large. Occasionally they may fill a major portion of the entire abdominal cavity and completely obscure the other organs.

Mesenteric cysts are most commonly found in the mesentery of the jejenum or ileum; the second most common location is in the mesentery of the sigmoid.[2] The cysts lie between the peritoneal leaves of the mesentery or omentum and most often tend to be situated close to the enteric border. Occasionally a cyst may extend out from either surface of the mesentery and partially surround the adjacent bowel in pantaloon fashion.

Such cysts may be encountered in patients of any age but are more apt to appear in childhood or early adulthood. Initially the only complaint may be merely the presence of a slowly enlarging abdominal mass. The progressive enlargement may continue for severeal years before being brought to medical attention. The most common symptom is pain of some nature, and this occurs in 80 per cent[2] of the patients. Nausea and vomiting may become associated with recurrent episodes of pain. Constipation is a less frequent complaint and has little diagnostic significance. Intestinal obstruction is a serious complication and may first call attention to the presence of a cyst. This occurs when the cyst impinges on the adjacent bowel and angulates it. Occasionally the cyst may be associated with a volvulus or intussusception.

Palpation may disclose the presence of a fluctuant mass with fairly well defined borders or the cyst may be so flabby as to defy palpation. Occasionally the cyst is so large and fluctuant that it suggests the presence of ascites. Moynihan described the characteristic transverse mobility of a mesenteric cyst with little vertical mobility.[9] Because of the absence of a pathognomonic sign or symptom these cysts are infrequently diagnosed preoperatively.

Roentgenograms may provide evidence of a homogeneous mass impinging upon or displacing some viscus. Occasionally calcification is present within the mass as reported by Vaughan.[11] Examination of the gastrointestinal tract with barium may show displacement of the viscera. Pyelography occasionally shows ureteral displacement by cysts of the mesocolon.

The methods of treatment of mesenteric cysts have consisted of aspiration, marsupialization, enucleation, and excision in continuity with the adjacent bowel. Enucleation, when possible, is the preferred treatment. It may be impossible to enucleate the cyst without impairing the blood supply of the adjacent bowel. Under such circumstances, excision of the cyst with bowel resection and end to end anastomosis may be performed. Currently the mortality from either of these procedures should be quite low. Aspiration and marsupialization have little to recommend them and should be avoided.

CASE REPORTS

Case 1. D. F. VUH 259134. This 7-year-old boy was admitted to the Vanderbilt University Hospital on July 23, 1956. Three years previously he had an episode of abdominal pain, nausea and vomiting. The examining physician at that time palpated an abdominal mass which he diagnosed as intestinal obstruction. This was treated by gastric intubation with subsidence of symptoms and the disappearance of the mass. Thereafter the boy experienced intermittent episodes of cramping, lower abdominal pain which were of short duration.

Two weeks before admission he fell, striking his abdomen. The abdominal wall turned "blue" the following day and he experienced more cramping, lower abdominal pain. He was nauseated but did not vomit. One week before admission he was admitted to another hospital. He was observed to be acutely ill with abdominal pain, pallid skin and a distended abdomen with a doughy mass in the lower portion. An abdominal operation was performed and a "cystic tumor containing dark fluid" was found with "loops of small bowel running in and out of the tumor." Nothing definitive was done. On the sixth postoperative day the child was referred to Vanderbilt University Hospital for further evaluation and treatment. His abdomen was moderately distended. A healing lower right paramedian incision was present. There was an ill-defined doughy mass occupying the entire lower abdomen. The mass was semi-fluctuant and non-tender. No movement of the mass could be elicited. On rectal examination there was an ill-defined diffuse mass anteriorly. Routine laboratory studies were normal.

A gastrointestinal series demonstrated displacement of the small bowel by a circular mass in the

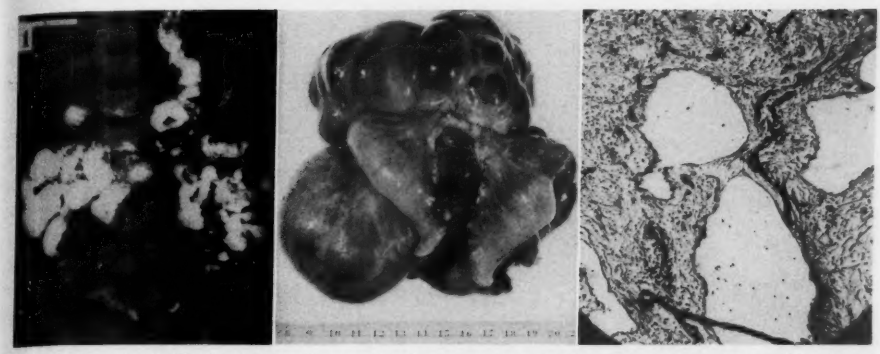

FIG. 1. Case 1. (*Left*) Gastrointestinal series demonstrating displacement of small bowel by homogeneous mass. (*Center*) Multiloculated cyst partially surrounding a loop of jejunum. (*Right*) Section of cyst composed of fibrous and smooth muscle tissue and inflammatory cells.

midabdomen, as is seen in figure 1 (left). There was no delay in the passage of barium through the small bowel. Intravenous urograms demonstrated a soft tissue density essentially in the midline overlying the lower lumbar vertebral bodies. There was prompt excretion of the medium in both renal collecting systems which did not appear remarkable.

At operation on July 26, 1956 the abdomen was opened through the previous incision. A large multiloculated cystic structure was found which was adherent to the parietal peritoneum by fibrinous adhesions. The cyst measured approximately 16 by 16 by 9 cm. and extended from the upper abdomen into the pelvic fossa. Following delivery of the cyst it was found that it had arisen between the leaves of the jejunal mesentery and almost completely enveloped a 25-cm. loop of jejunum in saddle fashion. The involved portion of jejunum was resected in continuity with the cyst and end to end open anastomosis was accomplished. An incidental appendectomy was performed. Postoperatively the child was afebrile and did well. He was discharged on the 6th postoperative day.

The tumor weighed 734 gm. and measured 15 by 15 by 6 cm. (fig. 1, *center*). The cyst was soft, fluctuant, multiloculated and had a blue hemorrhagic appearance. Some of the cystic areas measured 8 cm. in diameter. On cutting the specimen the walls measured from 1 to 2 mm. in thickness. The fluid was serous in nature, except in a few of the cysts it was blood tinged. The cysts did not communicate with each other. Microscopically the wall was composed of fibrous and smooth muscle tissue with a flattened endothelial lining (fig. 1, *right*). There was also some evidence of hemorrhage and chronic inflammation in the septa of the cyst.

Case 2. N. H. VUH 211150. This 35-year-old, colored female entered Vanderbilt University Hospital on August 16, 1956. A small mass in the left lower quadrant had been observed approximately eight years previously. This mass had increased in size gradually until one year before admission after which it enlarged fairly rapidly. She experienced occasional pain in the left lower lumbar area radiating to the left lower quadrant. Three weeks previously she noted anorexia, constipation and occasional vomiting. On examination, she was a well developed, well nourished, colored female in no distress. In the left lower quadrant of her abdomen there was a firm, smooth, round, slightly tender mass 12 cm. in diameter. The mass could be moved transversely but not vertically. Pelvic examination disclosed no abnormality. Urinalysis and routine blood studies were normal.

An intravenous urogram demonstrated a smooth ovoid soft tissue density approximately 12 by 14 cm. extending from the level of the 2nd lumbar vertebra to just below the crest of the ilium located on the left and overlying the lower pole of the left kidney. The left ureter was displaced somewhat medially in its midportion. A barium enema revealed displacement of the mid-descending colon anteriorly and medially (fig. 2, *left*).

On August 17, 1956, a laparotomy was performed through a left paramedian incision. A large, smooth cystic structure 14 cm. in diameter was present within the mesentery of the descending colon. The mass extended from the inferior pole of the left kidney to the broad ligament. The ureter was displaced medially by the mass. By incising the lateral leaf of the mesocolon this mass was easily enucleated. Postoperatively the patient had an uncomplicated course. She was discharged on the sixth postoperative day and subsequently has done well.

The specimen consisted of a unilocular cystic

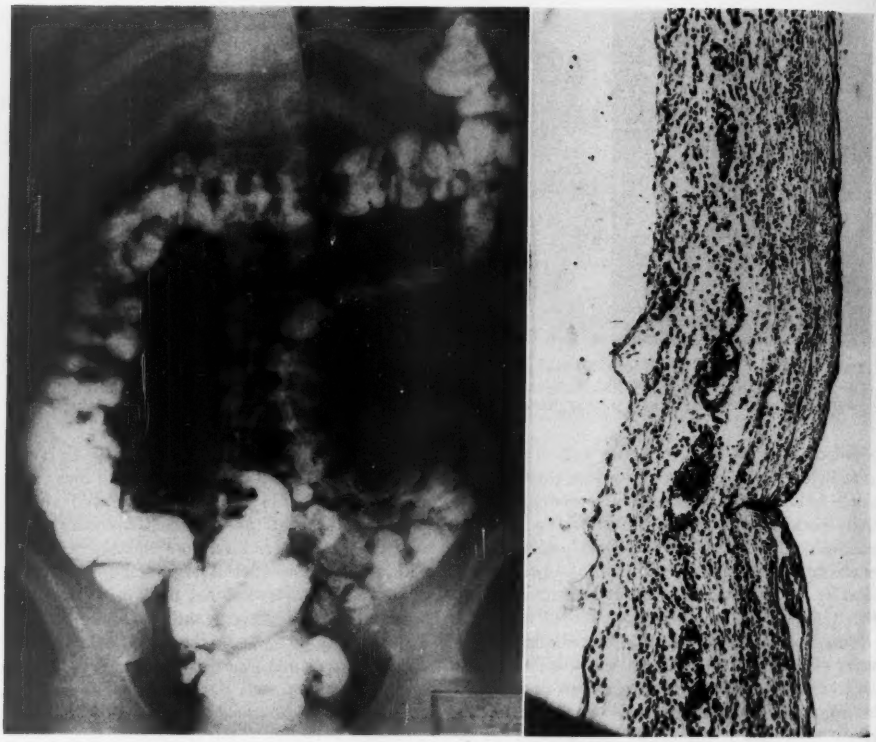

FIG. 2. Case 2. (*Left*) Barium enema examination showing displacement of descending colon by mesenteric cyst. (*Right*) Section of cyst wall consisting predominantly of fibrous tissue with lymphoid tissue and macrophages.

structure with an opaque appearance. It measured 12 cm. in diameter. Upon opening this cyst, clear serous fluid escaped. The cyst wall measured about 2 mm. in thickness and the inner lining was smooth and glistening.

Microscopically the cyst wall was composed predominantly of fibrous connective tissue which in areas was hyalinized and showed cholesterol clefts. Within the wall scattered accumulations of lymphocytes were seen. A few sparsely scattered epithelioid hemosiderin bearing macrophages were observed (fig. 2, *right*).

Case 3. M. L. VUH 263874. This 60-year-old white female entered Vanderbilt University Hospital on January 2, 1957 with pain in her abdomen. She had experienced intermittent, right upper quadrant abdominal pain for nine months and had noted abdominal distension intermittently. For three months before admission she had passed pencil stools. Rectal examination disclosed poor sphincter tone. Routine laboratory studies were within normal limits. Barium enema demonstrated a constricting lesion in the lower sigmoid. Sigmoidoscopy revealed a narrowing of the colon approximately 12 cm. above the anal orifice. Multiple biopsies failed to reveal evidence of carcinoma. Her abdomen gradually became more distended and a plain x-ray film revealed the large bowel to be markedly distended with gas down to the level of the proximal rectum. A long intestinal tube was inserted and on January 9, 1957 operation was performed through a left paramedian incision. A thin walled cystic structure measuring approximately 12 cm. in diameter was found within the transverse mesocolon. This cystic structure was unilocular and was enucleated by incising the anterior leaf of the transverse mesocolon. There was in addition marked narrowing of the lower sigmoid colon, which appeared to be inflammatory in nature. An anterior resection of the sigmoid and proximal portion of the rectum was performed with end to end anastomosis. Postoperatively the patient had no significant complications.

The cyst measured 12 cm. in diameter and had a smooth external surface; a large amount of thin, clear fluid escaped when it was opened. It was uni-

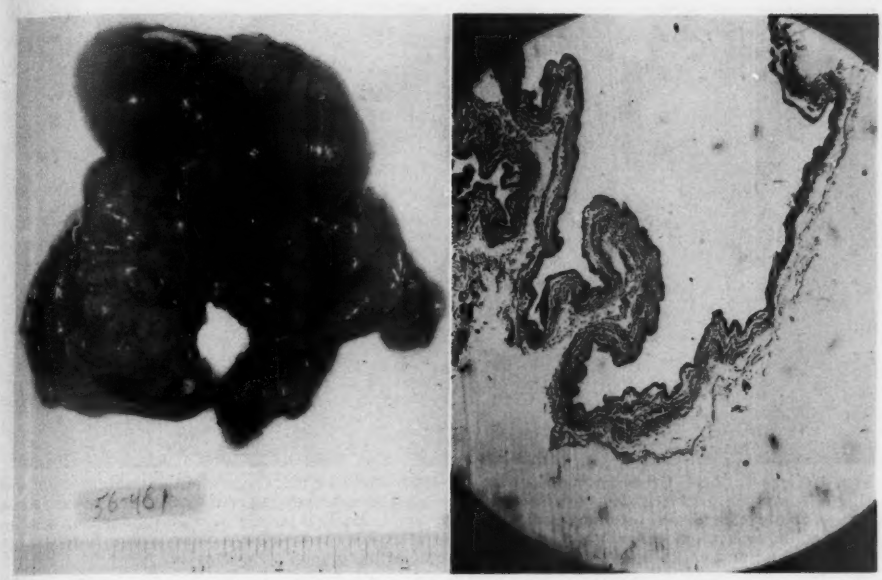

FIG. 3. Case 4. (*Left*) Excised multiloculated cyst partially enveloping a loop of ileum. (*Right*) Section of cyst wall showing flattened endothelial lining cells and loose connective tissue stroma.

locular and the inner walls were smooth and glistening. Microscopic sections of the wall revealed fibrous connective tissue in which there were interspersed a moderate number of lymphocytes. The inner lining was composed of flattened endothelium. The resected bowel showed changes compatible with diverticulitis and stricture formation.

Case 4. H. W. VUH 254107. This 6-year-old white male child was admitted to the Vanderbilt University Hospital on February 22, 1956 with abdominal pain. The child had been in good health until one day before admission when, after a normal bowel movement, he complained of mid-abdominal cramping pain which caused him to double up in bed. This cramping abdominal pain continued until the time of admission. He had one normal bowel movement shortly after the onset of the pain. He became nauseated and vomited once about six hours before admission. His temperature was 100.2 and his pulse was 180. The abdomen was scaphoid. There was some guarding in the lower abdomen. Tenderness was noted on deep palpation of the right lower quadrant just at the pelvic brim. There was no rebound or referred tenderness. Bowel sounds were normal. On rectal examination there was tenderness high on the right. Urinalysis was normal. The leukocyte count was 13,800 per cu. mm.

Operation was performed through a McBurney incision with the tentative diagnosis of appendicitis. As the peritoneum was opened a moderate amount of thin cloudy fluid escaped. The appendix appeared normal. Within the mesentery of the terminal ileum there was a multilocular cyst which measured 10 cm. in diameter. The cyst extended from the base of the mesentery to the enteric border and partially encircled the small intestine in a saddle fashion. It was felt that an incomplete obstruction existed. In addition, there were four distinct areas of ileo-ileal intussusception proximal to the cyst. All of these were reduced without difficulty. The mesenteric cyst was resected *en bloc* with the contiguous portion of ileum and an end to end open anastomosis was performed. Postoperatively the child did well without significant complications.

The specimen consisted of a protion of ileum which measured approximately 15 cm. in length (fig. 3, *left*). The serosal surface was smooth but purple in color. Within the mesentery of the ileum were approximately ten thin-walled, yellowish cysts of varying sizes. They appeared to be in intimate contact with the enteric border of the mesentery. The cysts ranged in size from 5 mm. to 3 cm. in diameter. The cysts were filled with thin serous fluid. Microscopically the walls were composed of a flat, endothelial lining and a very loose connective tissue stroma in which occasional chronic inflammatory cells and eosinophiles were seen (fig. 3, *right*).

Case 5. H. B. VUH 263099. A four-year-old white boy was admitted to Vanderbilt University Hos-

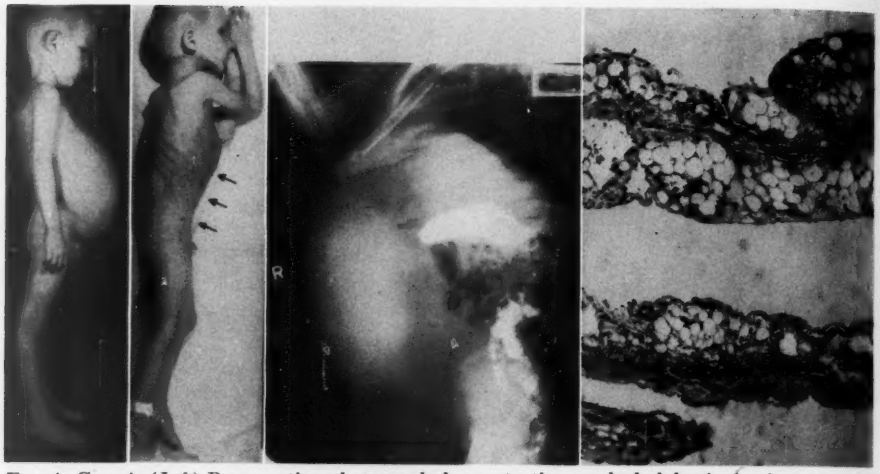

FIG. 4. Case 4. (*Left*) Preoperative photograph demonstrating marked abdominal enlargement with flaring of costal margins. (*Center left*) Postoperative photograph showing remarkable decrease in size of abdomen following excision of omental cyst. (*Center right*) Barium enema revealing posterior displacement of colon by homogeneous cyst. (*Right*) Section of cyst walls which are composed of fibrous tissue, collogen and a few smooth muscle elements with focal collections of lymphocytes.

pital on November 24, 1956, with a distended abdomen. At the age of 18 months abdominal enlargement was noted and had been progressive since that time. There had been no pain or symptoms referable to bowel function. One week before admission a paracentesis was performed and 500 cc. of serous fluid was withdrawn. Physical examination revealed a well developed and fairly well nourished white boy with marked abdominal swelling and flaring of the costal margins (fig. 4, *left*). A large fluctuant mass was present with borders extending well into the flanks. The remainder of the examination disclosed no other abnormality.

The routine laboratory studies were within normal limits. A plain x-ray film of the abdomen demonstrated a markedly enlarged abdominal space occupied by an evenly and symmetrically distributed homogeneous density. A barium enema revealed the colon to be displaced posteriorly by the large anterior mass (fig. 4, *center right*).

A laporotomy through a right paramedian incision was performed on November 28, 1956. An enormous cyst of the omentum was found which measured approximately 30 by 18 by 11 cm. The cyst appeared to be composed of two spaces and extended from diaphragm to pelvis, obscuring all other viscera. To facilitate its removal, 3900 cc. of serosanguineous fluid was aspirated from one of the loculations. Following this the cyst was easily removed in its entirety by excision of the greater omentum. No other abnormality was detected. His convalescence was uneventful and he was discharged on the fifth postoperative day.

Chemical analysis of the fluid removed from the cyst revealed the sodium to be 142 mEq./L.; potassium, 4.4 mEq./L.; chloride, 106 mEq./L.; and protein, 4.1 gm. per cent. The cyst had very thin, transparent walls with a septum dividing it into two loculations. The walls were smooth and glistening. Histologically the cyst wall was composed of fibrous tissue, collagen, and a few smooth muscle elements (fig. 4, *right*). On the external surface it was covered by mesothelial cells which appeared slightly swollen. The internal lining consisted of very flattened endothelial cells. Some inflammatory cells were present, principally eosinophils and vacuolated macrophages. A few collections of lymphoid tissue were present within the wall.

DISCUSSION

In each of the four cases of mesenteric cyst, pain was a prominent symptom. In one of the patients the pain probably was related to the presence of unassociated, partial obstruction of the large bowel. Handelsman and Ravitch[6] have attached considerable importance to the occurrence of pain in children with mesenteric cysts. Burnett and associates, in a collected series of 71 patients, found that pain of some nature was present in 81 per cent.[2] It would appear, however, that unless intestinal obstruction occurs the pain is rather vague and has no characteristic feature. The chief complaint of the patient with the omental cyst was progressive and painless enlargement of the abdomen. This was so impressive

that paracentesis had been performed with the mistaken assumption that it represented ascites.

A palpable mass was present in two of the patients with mesenteric cysts and in the patient with an omental cyst, which made possible an accurate pre-operative diagnosis. In all three patients the mass was typically fluctuant and the cyst of the mesocolon demonstrated the usual transverse mobility to palpation. In one of the children, the finding of a mass was probably obscured by the acute abdominal condition caused by intussusception of the ileum. It is possible that the presence of the mesenteric cyst could have initiated the intussusception. In one patient the presence of a mesenteric cyst was an unexpected and incidental finding at operation for an unrelated condition.

In three of the five patients in whom roentgenologic study was performed there was a definite abnormality caused by the presence of the cyst. This was manifested as the presence of a homogeneous mass with compression or displacement of the adjacent viscera. In case 3 the cyst had not reached sufficient size within the relatively mobile transverse mesocolon to cause compression.

The mesenteric cysts in all patients were relatively large and the omental cyst occupied the major portion of the abdominal cavity. The omental cyst was bilocular. Two of the mesenteric cysts were multilocular and were located in the mesentery proper while the two unilocular cysts were present within the mesocolon. This concurs with Moynihan's original concept regarding the location of unilocular and multilocular cysts.[9] The reason for this particular characteristic is not clear.

The fluid in all of the cysts was serous in nature except in cases 1 and 5 in which a few of the cystic spaces were filled with bloody fluid. This undoubtedly was related to the antecedent trauma and previous operation. In none did the fluid have the typical appearance of chyle. Chemical examination of the fluid from the omental cyst revealed the electrolyte and protein concentrations to be similar to that of plasma.

The cystic walls consisted essentially of endothelial and connective tissue elements. The finding of well defined smooth muscle in one of the mesenteric cysts (case 1) and in the omental cyst is not an unusual observation and should not cause confusion with intestinal duplications. Moore[8] found smooth muscle present in all four of his cases and Handelsman and Ravitch[6] found it present in three of their four cases. In none of the cysts were there histologic elements suggesting a urogenital origin.[7]

When feasible, simple excision or enucleation is the preferable procedure. If excision compromises the vascular supply of the bowel, local resection of the intestine and cyst with primary end to end anastomosis should be performed.

SUMMARY

Mesenteric and omental cysts are rare abdominal tumors which may be seen in patients of almost any age. The etiology of the cysts remains obscure. There is no characteristic symptomatology although pain of some nature is frequently present. The roentgenologic abnormalities are usually caused by compression of adjacent viscera. Microscopically the cysts are composed of endothelial and connective tissue elements.

Four cases of mesenteric cysts and one case of an omental cyst are described. All were subjected to surgical therapy with recovery. In two cases of the mesenteric cysts and in the case of the omental cyst, this was accomplished by simple excision while in the other two mesenteric cysts it was necessary to resect the adjacent bowel in continuity with the cyst. All patients recovered satisfactorily.

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PILOT STUDIES IN THE TREATMENT OF BURNS IN INFANTS AND CHILDREN*

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Since 1951 the author has treated 57 infant and child burns and in that time a number of problems have arisen with regard to their treatment. There is very little literature available to pinpoint the best therapeutic approach for the care of these small patients. A number of authors have devised formulas which have worked well in their hands but when used by us have not proven satisfactory. As tolerances to electrolytes, colloids or water are stated to be so much less in infants and children than in adults, a number of questions arise as to the relative amounts of these constituents which should be supplied and the proper time of their administration. Additional factors are concerned with the relative importance of endocrine factors, in particular with the adrenals in the regulation of salt and water metabolism. The author feels that if the physiologic principles of the pathology of burn are moderately well recognized, it will be difficult to harm even a child from over treatment or under treatment. These principles have been adequately covered in the literature.

In 1954, a research program was begun which included daily blood chemistries and complete 24-hr.-urine studies over a period of seven days for each infant and child under 12 years of age with a greater than 20 per cent burn. To date, nine children have been thus investigated. Total sodium, potassium, chloride, nitrogen and corticoids were determined. Hourly intakes and outputs were recorded for the first three days and at eight hourly intervals thereafter. All foods were weighed and the intake of sodium, potassium, nitrogen, proteins and calories were computed.

METHODS OF TREATMENT

Of the nine patients studied, two were given a balanced electrolyte solution (5 per cent dextrose in Ringer's lactate) for 48 hr. with blood as a colloid support, as advocated by Fox[6] and Wilson[12] (tables 1 and 2). Two received less or nearly the amount of sodium advocated by the Brooke Army Formula[7] (see tables 3 and 4). The remainder (tables 5, 6, 7, 8 and 9) received considerably more sodium than advocated by the Brooke formula yet not nearly as much as recommended by Fox and Wilson. After admission to the hospital, no patient was given any electrolyte-free solution intravenously during the first 48 hr. All cases were started on 5 per cent dextrose in Ringer's lactate as their initial therapy. Cases 5 to 9 were started initially on dextrose in Ringer's lactate and, following establishment of their urinary output, a lesser concentrated sodium solution was prescribed. This was prepared by mixing 5 per cent dextrose in Ringer's lactate with 5 per cent glucose in water, the concentration of sodium being made less following initial Ringer's lactate therapy, and still less in the second 24 hr. In early cases, blood was used as colloid support but in later cases plasma was used. In the latter group, small amounts of blood were given near the end of the first 24 hr. In all but case 6, plasma was given later in the burn period to support low plasma proteins. The following are reviews of the patients studied: (It should be remembered that the normal hematocrit in the child is 35).

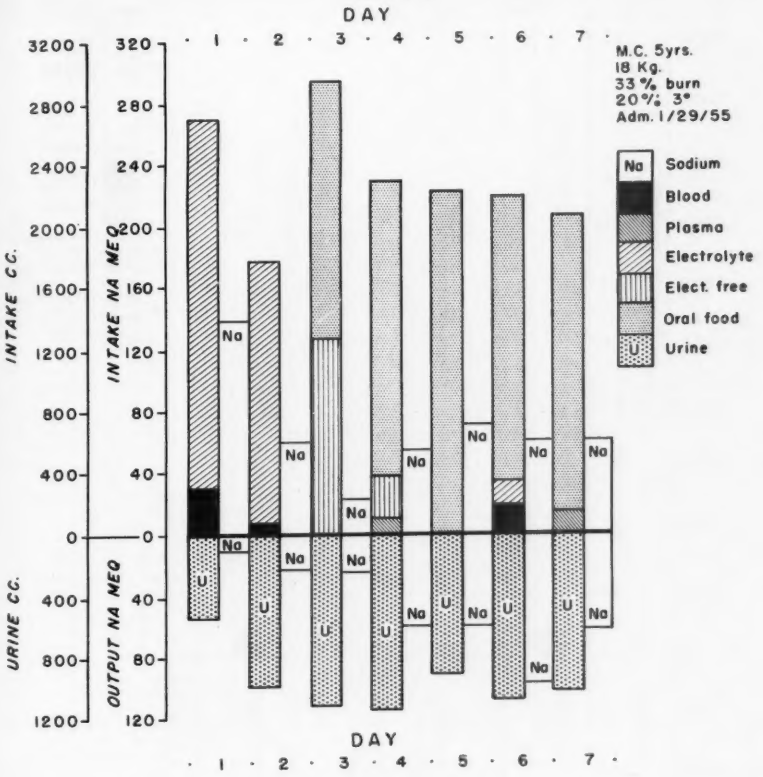
CASE REPORTS

Case 1 (table 8). D. H , was a 24-kg., 7-year-old male child admitted January 10, 1956, with a 23 per cent burn, of which 13 per cent was third degree. He was treated by administration of dextrose in Ringer's lactate for the first 48 hr. He was given 100 cc. of blood at the 14th hr. and 200 cc. of blood at the 20th hr. Because of low proteins, he was given 75 cc. of plasma at the 77th and 92nd hr. He was started on supplemental potassium chloride on the 6th day. Because of his low hematocrit at the 96th hr. he was given 125 cc. of blood. In spite of this large sodium intake, his serum sodium did not become greatly elevated. Ten per cent invert sugar was given slowly after the 48th hr. The only

* This work was supported in part by U. S. Public Health Grant No. H-1898 from Department of Surgery, San Antonio Postgraduate Division, University of Texas.

† Associate member Southwest Research Foundation.

TABLE 1

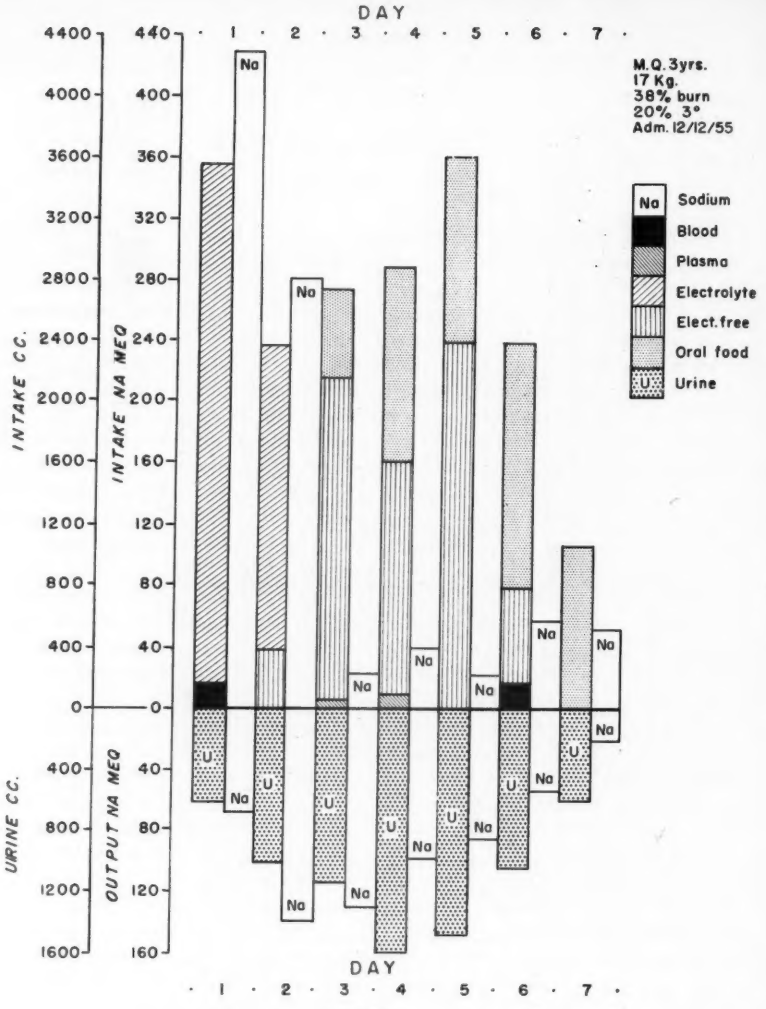


CHEMISTRIES	Adm.	24	48	72	96	120	144
HCT	42	54	54	52	36	30	36
CO_2	29	38	21	22	21	18	22
Na	137	131	132	130	146	142	144
Cl	90	88	83	82	103	96	106
K	6	6.8	6.6	6.1	6.4	5.1	5.7
NPN		50		34			
T.Pro		4.8	5.4	5	3.1	5.1	4.9
Alb.	2.4	3.9	3.5	1.8	2.6	2.4	2.4
Glob.	2.4	1.5	1.5	1.3	2.5	2.6	2.5
pH		7.32	7.3	7.35	7.35	7.45	7.45

sodium intake following this period was in the form of oral foods and saline given with plasma or blood. There was little sodium excretion during the first 24 hr. but considerable quantities thereafter, and he stabilized the intake and output of sodium by the 6th day. No blood pressures were recorded. Clinically, he was much more edematous than other cases treated with lesser amounts of sodium but at no time was there a significant change in vital signs.

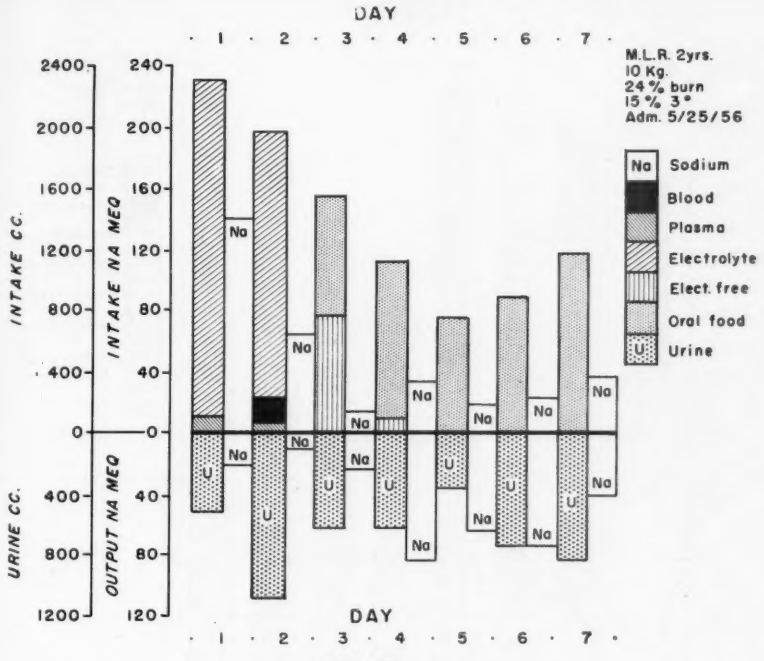
Case 2 (table 2). M.Q., was a 17-kg., 3-year-old female child admitted December 12, 1955, with a 38 per cent burn, 20 per cent of which was third degree. She was treated with dextrose in Ringer's

TABLE 2



CHEMISTRIES	Adm.	24	48	72	96	120	144
HCT	45	47	42	37	35	32	36
CO_2	16	22	21	23	24	23	24
Na	149	160	156	150	146	146	147
Cl	110	118	105	105	103	104	105
K	5.2	6.7	5.8	5	4.9	4.3	5.2
NPN	52	25	40	14			
T.Pro.	4.7	3.04	3	5.08	5.09	4.6	4.5
Alb.	2.5	2	1.08	3.25	3.78	3.1	2.3
Glob.	2.2	1.04	1.92	1.83	1.3	1.5	2.2
pH							

TABLE 3



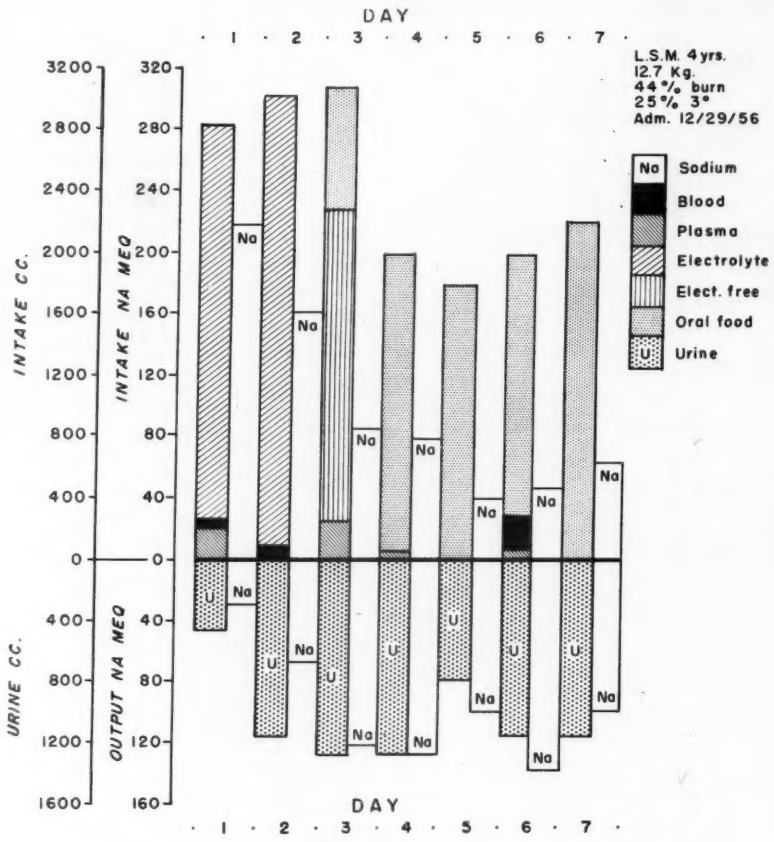
HOURS

CHEMISTRIES	Adm.	24	48	72	96	120	144
HCT	35	31	44			33	
CO_2	19	16	18				
Na	163	160	146			140	
Cl	108	100	100			102	
K	5.6	7.2	5.4			6.6	
NPN	65	25	25			40	
T. Pro.	4.5	4	4.6			4.8	
Alb.	2.7	2.7	2.8			2.5	
Glob.	1.8	1.3	1.9			2.4	
pH							

lactate for 48 hr. She was given 150 cc. of blood at the 9th hr. Because of her low serum proteins she was given 50 cc. of plasma at the 41st hr. and 100 cc. of plasma at the 82nd hr. In view of a low hematocrit she was given 150 cc. of blood at the 126th hr. As seen in the table, she received 430 mEq. of sodium the first 24 hr. and 280 mEq. the second 24 hr. She was not as edematous as the first case. It will be noted that she had an extremely low total protein at the 24th hr., having received only blood as colloid support. No blood pressures were recorded. Her sodium excretion and intake of sodium were stabilized by the 6th day. Potassium was added to her diet on the 5th day.

Case 3 (table 7). J. E. H., was an 18.6-kg., 5-year-old female, admitted January 8, 1957, with a 35 per cent total burn of which 20 per cent was third degree. This child was admitted 18 hr. postburn, having received during the interim only one and a half quarts of water and a bowl of soup. Through a misinterpretation of orders she was given even a less concentrated sodium solution than was planned. She was administered a solution containing 52 mEq. of sodium per L. in 5 per cent

TABLE 4

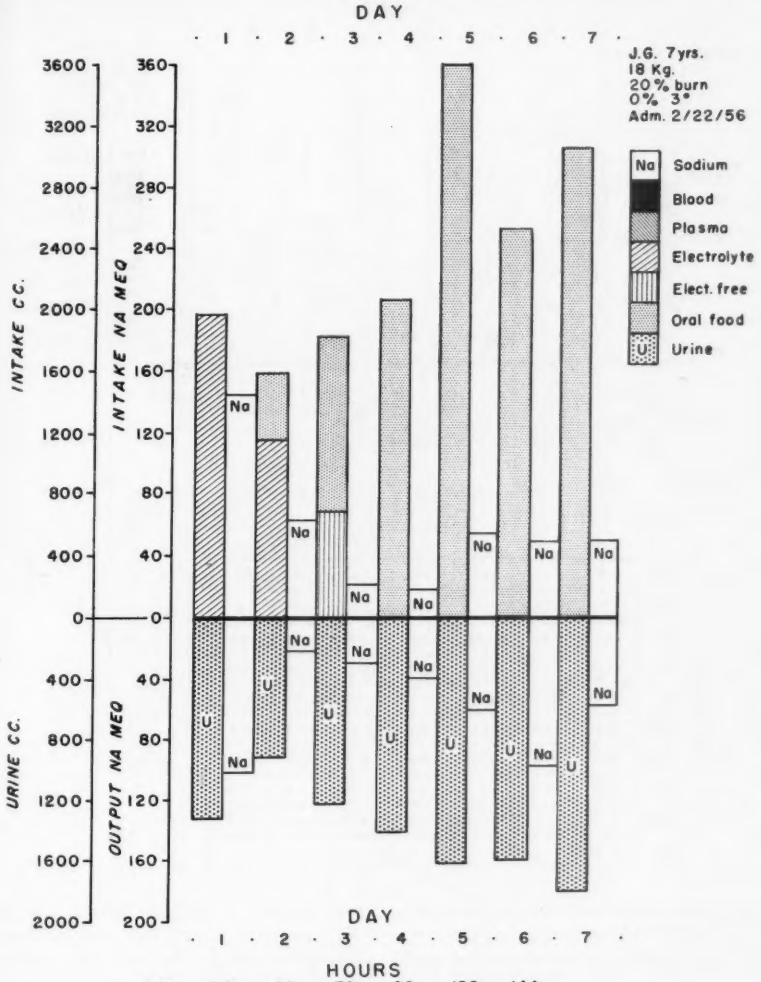


HOURS

CHEMISTRIES	Adm.	24	48	72	96	120	144
HCT	50	54	50	49	33	29	30
CO_2	14	16	16	16	19	21	21
Na	154	140	140	151	146	130	140
Cl	107	102	103	104	103	100	100
K	7.5	5.8	6.3	5.3	5	4.5	4.2
NPN	29	50	50	24	39	42	26
T.Pro.	5.6	4.5	4.2	4.5	5.2	5.2	4.8
Alb.	4.2	2.9	2.7	2.9	3	3	3
Glob.	1.3	1.6	1.5	1.6	2.2	2.2	1.8
pH	7.3	7.4	7.45	7.45	7.45	7.45	7.45

glucose. This was obtained by mixing 400 cc. dextrose in Ringer's lactate with 600 cc. of 5 per cent dextrose in water. This was continued until the end of the 48th hr. Blood pressures taken hourly revealed the patient to be normotensive throughout. She was given 150 cc. of plasma at the 44th hr. and 100 cc. of blood at the 50th hr. It was felt that she should have received more sodium and we were

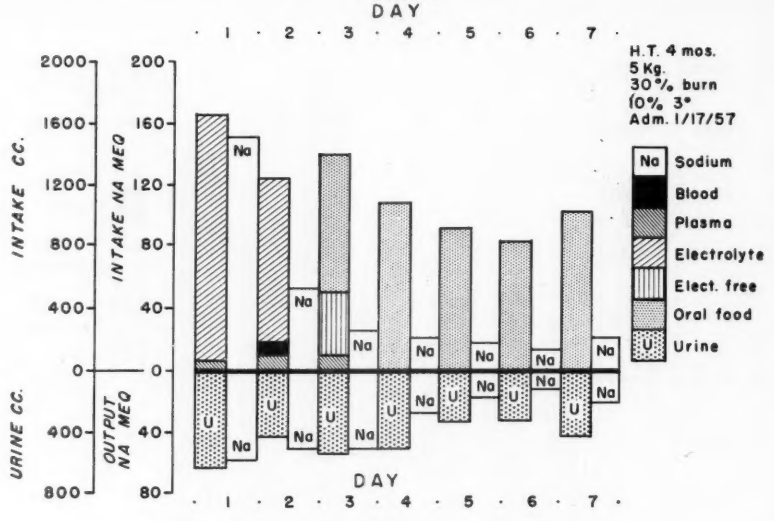
TABLE 5



HOURS

		Adm.	24	48	72	96	120	144
CHEMISTRIES	HCT	43	46	45	45	43	42	42
	CO_2	23	21	22	21	24	25	25
	Na	140	144	144	145	145	140	144
	Cl	105	111	107	113	111	105	111
	K	5	6.8	6.8	7.7	6	6	7.3
	NPN	32	35	29	28	25	25	25
	T. Pro.	7.2	6.4	5.9	5	6.7	6.3	6
	Alb.	4	4.2	3.9	3.4	4.2	3.6	3.5
	Glob.	3.2	2.2	2	1.6	2.5	2.7	2.5
	pH							

TABLE 6



HOURS

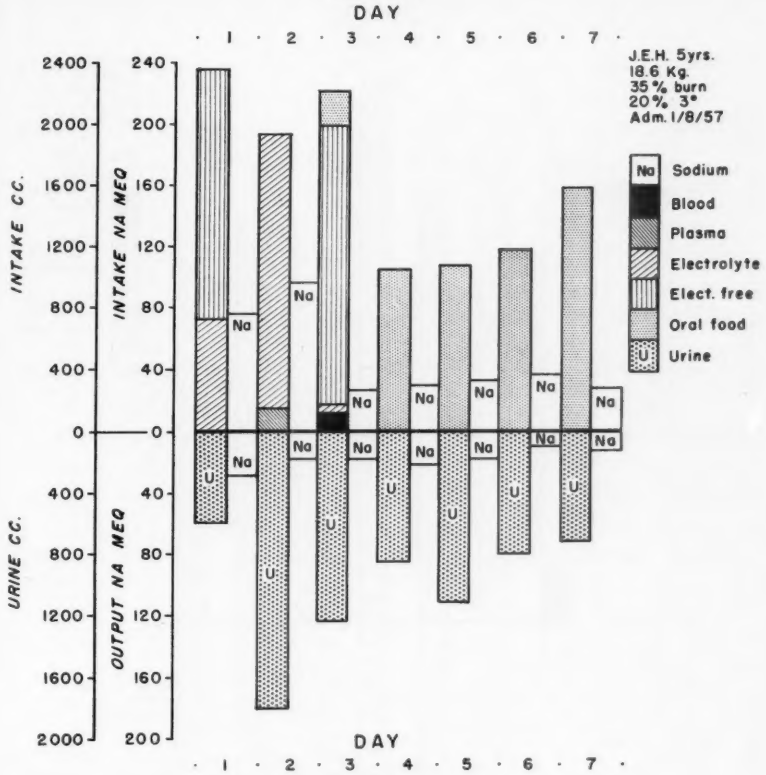
CHEMISTRIES	Adm.	24	48	72	96	120	144
HCT	38	32	33	32	35	38	38
CO_2	15	25	16	20	20	15	15
Na	141	140	140	151	145	140	138
Cl	102	100	101	102	102	100	100
K	6.4	5.2	4.5	6.1	6	6	6
NPN	59	39	45	42	56	56	64
T.Pro.	4.3	4.6	5.7	5.9	6.2	6.6	6.3
Alb.	3.6	3.4	3.4	3.7	3.7	4.4	3.6
Glob.	1.3	1.2	2.3	2.2	2.4	2.3	2.7
pH	7.42						

in a quandary as to whether to continue electrolytes past the 48th hr. but as the serum sodium was normal at that time, no more was given. She should have received plasma shortly after admission but we decided to wait and see if there was any hypotensive phase before administering it. There was none. As may be seen in table 3 there was very little sodium excretion during the study period and even at the 7th day there was a greater intake than output. She did not suffer any serum protein drop during the 7 days studied.

Case 4 (table 9). F. Y., was a 10-kg., 3-year-old female, admitted January 10, 1956, with a 28 per per cent total burn, of which 7 per cent was third degree. She was started on 75 cc. dextrose in Ringer's lactate until her urinary output became adequate; then was placed on a solution containing 32.5 mEq./L. of sodium obtained by mixing one part dextrose in Ringer's lactate with three parts of dextrose in water. This was continued to the end of the 26th hr. She was then put on a sodium solution containing 21.5 mEq. per L. Ringer's lactate and dextrose in water 1:5. She was given 100 cc. of blood at the 32nd hr. As may be seen in table 4, she had a low serum protein on the 2nd and 3rd days and was given 50 cc. of plasma at the plasma at the 76th hr. She had a low hematocrit at the 96th hr. and was given 200 cc. of blood. No blood pressures were taken. She was placed on supplemental potassium the 6th day. This case was treated early in our studies in an attempt to find a proper mixture of sodium for these burn patients. We feel that she should have received more sodium the second day as her output of it was quite low that day.

Case 5 (table 1). M.C., was an 18-kg., 5-year-old

TABLE 7

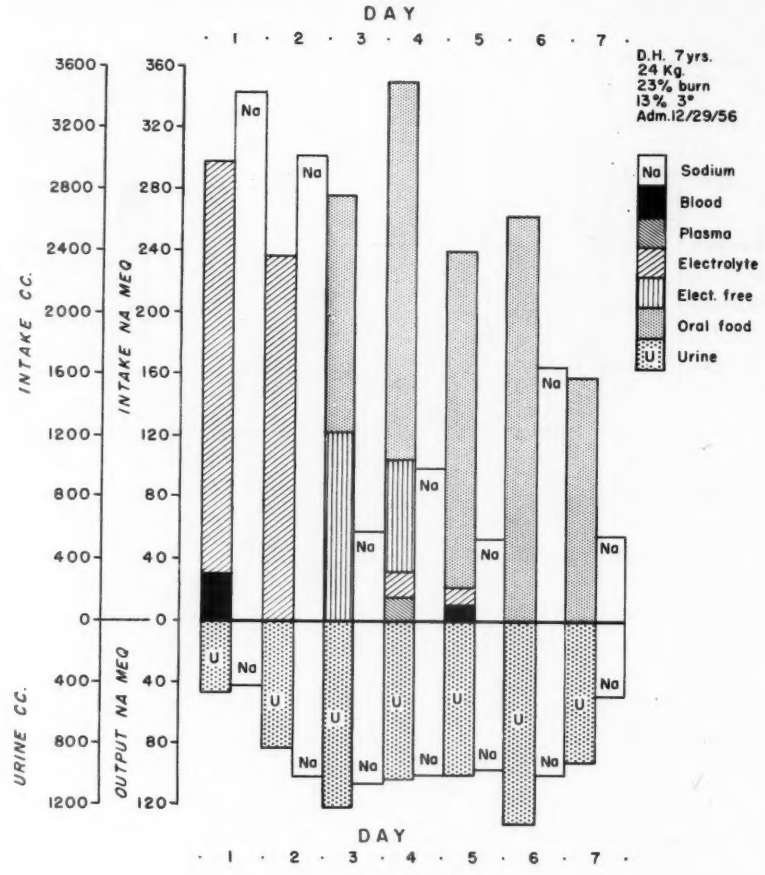


HOURS

CHEMISTRIES	Adm.	24	48	72	96	120	144
HCT	50	45	38	36	40	39	39
CO_2	20	22	24	24	24	24	30
Na	140	143	138	140	140	140	135
Cl	100	101	98	99	100	100	92
K	6.6	6.2	5.8	4.8	5.3	5.6	5.7
NPN	59	39	60	35	31	31	35
T. Pro.	5.4	5.7	5.4	5.3	5.4	5.4	5.4
Alb.	3.2	2.9	3.4	3	3	3	3.03
Glob.	2.2	2.8	2	2	2.4	2.4	2.39
pH	7.42	7.45	7.45	7.45	7.45	7.45	7.45

female, admitted November 29, 1955, with a 33 per cent total burn of which 20 per cent was third degree. She was given 250 cc. dextrose in Ringer's lactate before an adequate urinary output was established; then was placed on a sodium solution containing 43 mEq. per L. (Ringer's lactate and dextrose in water 1:2) until the 36th hr.; then a solution containing 32.5 mEq. per L. (Ringer's lactate and dextrose in water 1:3) until the end of 48 hr. Two hundred seventy cc. of blood were given during the first 24 hr. and 45 cc. of blood the second 24 hr. She was supported later with blood and plasma. This child developed extremely low plasma proteins the 4th day. Supplementary

TABLE 8

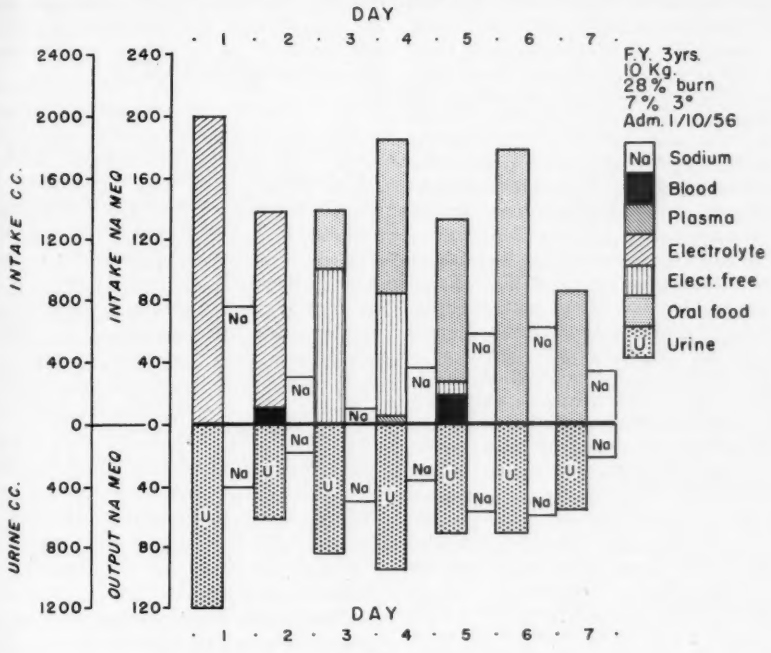


CHEMISTRIES	Adm.	24	48	72	96	120	144
HCT	45	46	42	36	30	38	38
CO_2	14	21	23	25	21	27	26
Na	145	145	145	145	148	140	144
Cl	103	105	105	105	107	110	112
K	5.6	6	5	5	5.5	5.7	5
NPN	58	58	42	31	30	30	30
T.Pro.	5.8	6	4	3.9	4.8	4.5	4.5
Alb.	4.5	4.5	2.8	2.7	2.8	2.7	2.7
Glob.	1.3	1.6	1.2	1.2	2	1.8	1.8
pH	7.45						

potassium was started on the 5th day. No blood pressures were taken. It is felt that she received too little sodium during the first 2 days as her sodium output was quite low during the first 72 hr.

Case 6 (table 5). J. G., was an 18-kg., 7-year-old male, admitted February 22, 1956, with a 20 per cent second degree burn. He was given 550 cc. of dextrose in Ringer's lactate; then a sodium solu-

TABLE 9



HOURS

CHEMISTRIES	Adm.	24	48	72	96	120	144
HCT	34	46	42	34	29	36	40
CO_2	16	17	20	21	23	23	23
Na	155	152	150	150	155	146	143
Cl	120	126	118	118	119	113	112
K	6.6	7.8	5.5	5.5	6	6.2	6.5
NPN		48	42	28	28	28	
T.Pro.	6.6	5.8	4	4	4.9	5.4	5.4
Alb.	4.6	4.6	2.8	2.7	2.7	3.4	3.4
Glob.	2	1.2	1.2	1.3	2.2	2	2.0
pH	7.5			7.55			

tion containing 65 mEq. per L. (Ringer's lactate and dextrose in water 1:1) until the end of 12 hr.; then a sodium solution containing 43 mEq. per L. (Ringer's lactate and dextrose in water 1:2) until the end of 24 hr.; then a sodium solution containing 26 mEq. per L. (Ringer's lactate and dextrose in water 1:4) until the end of 48 hr. No blood or plasma was given. No blood pressures were taken. This is an unusual case in that the patient excreted 100 mg. of sodium the 1st day. Since his burn was not great the relatively high sodium intake possibly caused a marked suppression of aldosterone allowing him to excrete his excess sodium so rapidly. He stabilized his sodium intake and output on the third day.

Case 7 (table 4). L. S. M., was a 12.7-kg., 4-year-old female, admitted December 29, 1956, with a 44 per cent total burn of which 25 per cent was third degree. She was given 600 cc. of dextrose in Ringer's lactate; then a sodium solution containing 78 mEq. per L. (Ringer's lactate and dextrose in water 6:4) until the end of 24 hr.; then a sodium solution containing 52 mEq. per L. (Ringer's lactate and dextrose in water 4:6) until the end of 48 hr. Blood pressures were checked each hour and she developed a hypotension at the 8th to the 10th

hr. Plasma infusions brought the blood pressure to normal and it remained so thereafter. She was supported with 100 cc. of plasma at the 5th hr. and 75 cc. of plasma at the 9th hr. She was given 75 cc. of blood at the 17th hr. and 100 cc. at the 33rd hr. On the 3rd and 4th days she was again supported with plasma. She was given blood the 6th day because of a low hematocrit. It was felt that she was treated adequately. Her serum proteins did not drop. She was stable on her intake and output of sodium by the 7th day. She received no supplemental potassium.

Case 8 (table 6). H. T., was a 5-kg., 4-month-old male baby admitted January 17, 1957, with a 30 per cent burn, of which 10 per cent was third degree. He was given 500 cc. of dextrose in Ringer's lactate before an adequate urinary output could be established; then was put on a sodium solution containing 78 mEq. per L. (Ringer's lactate and dextrose in water 6:4) until the end of 24 hr. then a sodium solution containing 52 mEq. per L. (Ringer's lactate and dextrose in water 4:6) until the end of 48 hr. He was given 75 cc. of plasma the first 48 hr. and 75 cc. of blood. His serum proteins did not drop. We feel that this infant probably received too much sodium as we had to give him so much solution initially to establish his urinary output. However, this case also proves the ability, even of infants, to excrete adequately their excess sodium if it is given early in the treatment of the burn. This infant's blood pressure remained normotensive until the 59th hr.; then he developed a hypertension with systolic pressure above 200. This hypertension persisted for several weeks before returning to normal. This phenomenon has been reported elsewhere.[7]

Case 9 (table 3). M. L. R., was a 10-kg., 2-year-old female, admitted May 25, 1956, with a 24 per cent total burn, of which 15 per cent was third degree. She received 185 cc. of dextrose in Ringer's lactate; then was put on a sodium solution containing 65 mEq. per L. (Ringer's lactate and dextrose in water 1:1) until the 24th hr.; then a sodium solution containing 26 mEq. per L. (Ringer's lactate and dextrose in water 1:4) until the end of 48 hr. Blood pressures were taken each hour although at the time the significance of low blood pressures was not fully appreciated. She stayed in a hypotensive state for 18 hr. When it became apparent that increasingly larger quantities of fluid were necessary to insure a minimal urinary output, the chart was reviewed and it was noted that her blood pressure had not been up to 100 systolic after admission. She was immediately given 100 cc. of plasma and became normotensive. Her systolic pressure again fell on the 26th to 28th hr. and she was given an additional 50 cc. of blood at the 29th hr. Because of prolonged hypotension she failed to excrete appreciable sodium until the 4th day. She was extremely edematous until the 5th day. Because of a breakdown of liaison with the laboratory no chemistries were performed on the 3rd, 4th and 6th days.

DISCUSSION

Although it is not shown on the tables, intake and output of potassium were studied. During the early study period supplemental potassium was started on the 5th and 6th days. In the later cases no supplemental potassium was given. As seen by the tables, some of the serum potassium levels were rather high during the early phase of the burn but the cases seemed to suffer no ill effects from this. There were no cases suffering from hypokalemia whether or not potassium was added to the diet. In this series, though not indicated on the tables, there was no particular correlation between the extent of the burn, the degree of the burn and the amount of potassium excreted. It was noted, however, that the younger the child, the less the potassium excretion. It is our opinion at the present time that supplemental potassium added to the feeding program is unnecessary provided the patient is eating well, as most foods contain a high amount of potassium.

In reviewing the cases, the reader may be impressed by the wide variety of forms of treatment these young individuals can receive and still survive. It was with this thought in mind that the author decided to study a series of cases and by reviewing them, both from a clinical and laboratory standpoint to determine what is helpful and what is harmful to the patient in the early phase of the burn. We also wished to ascertain, if possible, an easier and more foolproof method of treatment.

We believe we have determined that the infant's and child's tolerance to excess sodium during the first 48 hr. is great. The treatment advocated by Fox, Wilson *et al.*, was employed in patients 1 and 2. We do not feel that this is an ideal form of treatment. Even though these two patients survived, they were more edematous and retained their edema longer than did the other children treated. Darrow states that rehydration occurs, except with marked salt loss, much more easily and quickly when solutions are given which contain less salt than does physiologic saline; also, severe potassium deficiency can occur when physiologic saline is used.[5]

We feel that a more rational approach pro-

vides for the administration of a balanced electrolyte solution such as dextrose in Ringer's lactate initially when the losses are so rapid and great;[3, 4, 8] then, to give a less concentrated sodium solution after the urinary output has been established, obtained by mixing dextrose in Ringer's lactate with dextrose in water. This is not an idea original with the author. Talbot,[11] Alway[1] and Darrow[5] have advocated its use for maintenance following initial hydration with an isotonic solution. Pediatricians utilize it routinely in their treatment of diarrheas. It is our opinion that stored plasma is the colloid of choice as it is the type of colloid lost in the wound edema, and we have observed that it will promptly restore to normotension a patient who has been hypotensive. Since its loss is early, it should be given early. Prophylactically, we believe that it should be administered within the first 4 or 5 hr. postburn and repeated within the first 16 hr. postburn. A small amount of blood near the end of the first 24 hr. is then employed. The amount of total colloid used may be based on the following formula:

Kg. body weight × per cent third degree or deep second degree burn × 1

Plasma or blood should be administered in a separate vein, if possible, to allow continual administration of electrolyte solution; otherwise, the urinary output will drop during the colloid infusion. We feel strongly that blood pressure recordings each hour are extremely important whenever it is possible to obtain them; that more patients die from the consequences of shock than from electrolyte imbalance and that whenever a patient becomes hypotensive he should receive plasma or blood. It has been our observation that 75 cc. of plasma administered to the infant and 150 to 200 cc. to the child should restore the blood pressure to normal. Blood pressures are easier to determine than may be expected with a burned upper extremity. There is usually a small area of unburned skin in the cubital fossa. A sterile towel can be wrapped around the arm if it is burned and the pressure cuff wrapped around the towel. Plasma may be helpful at the 3rd or 4th day when the serum proteins fall very low in these children. The author, through bitter experience, must admonish that at this point plasma should be given sparingly and only if the total proteins are low; as it, like sodium and water, is being mobilized back into the blood stream at this time, and there is danger of hypervolemia developing if too much plasma is given. We have seen no case of homologous serum jaundice from the use of stored plasma. It is our opinion that the infant's output should be between 25 and 35 cc. per hr. No particular harm ensues from an excessive or diminished output over a 1- to 2-hr. period, but it should alert the attending physician to change his treatment. Excessive output merely means that too much fluid has been administered. Diminished output may signify several things: inadequate intake, hypotension, hyponatremia or, particularly in the infant, a plugged catheter. The water tolerance test (rapid infusion of 5 per cent dextrose in water) for oliguria, which is advocated by some authors[2, 10] may be dangerous. If the patient is suffering from hypotension or hyponatremia, a rapid infusion of dextrose in water may cause death. In the event it is impossible to check the blood pressure, hypotension can be suspected if after the urinary output has been established, it becomes evident that increasingly larger hourly quantities of intravenous fluids are necessary in order to obtain a minimal urinary output. In this event, the patient is probably in shock and a colloid should be given. If shock can be ruled out for oliguria, the electrolyte solution being administered is simply increased. In all cases of anuria or oliguria, particularly in the infant, we should determine the patency of the catheter.

We allow nothing by mouth for 48 hr. postburn. There are many reasons for this, the most important one being that we can more precisely regulate the intake and thereby the output. We give no intravenous sodium solution after 48 hr. unless the serum sodium is below 130 mEq. per L. and in no instance have we seen this where we have handled the early treatment. At the 48th hr. we start hourly feedings and continue with 10 per cent invert sugar in water intravenously. This intravenous feeding is continued until the patient is able to take adequate food and fluids orally. We stop concerning ourselves with precise hourly outputs of urine after the 48th hr.

We obtain plasma proteins daily after the first 48 hr. and, if they are low, support them with small plasma infusions until they start rising.

Schlegel[9] has offered some novel ideas on the treatment of shock and severe stress, stating

that where large quantities of fluid are necessary, overhydration is common and sodium must be administered in quantity to bring the sodium level prior to stress. He further states that increased adrenal activity seems to render the organism more capable of handling a sodium load provided that a sufficient initial amount of sodium in excess of the concentration in the serum has been given under such conditions.

From the results of our studies, we feel that the initial treatment of the massive burn, whether in the infant, child or adult, should be dextrose in Ringer's lactate and that this administration should be continued until an adequate urinary output has been established, maintaining hourly checks on the blood pressure and administering plasma both prophylactically according to the formula mentioned previously, and for hypotension. The amount of early, hourly intravenous intake necessary for the child will depend on the size of the patient and the extent of the burn. For the larger child with a more extensive burn 250 cc. per hr. may be required early. For the small child, or the less extensive burn, 100 cc. to 150 cc. per hr. may be adequate.

Our present ideas for the treatment of burns of over 30 per cent of the body surface consist of the following:

Initial administration of 5 per cent dextrose in Ringer's lactate until an adequate urinary output has been established. Following this, a mixture of dextrose in Ringer's lactate and dextrose in water should be given. A solution containing 78 mEq. of sodium per L. (600 cc. dextrose in Ringer's lactate mixed with 400 cc. dextrose in water) is administered until the 24th hr. From the 24th to the 48th hr. a lesser concentrated sodium solution containing 52 mEq. of sodium per L. (400 cc. dextrose in Ringer's lactate mixed with 600 cc. dextrose in water) is given. After 48 hr. 10 per cent invert sugar in water is administered slowly and oral feedings are allowed.

For burns under 30 per cent, dextrose in Ringer's lactate is given initially until the urinary output has been established; then, a solution containing 52 mEq. of sodium per L. (400 cc. dextrose in Ringer's lactate mixed with 600 cc. dextrose in water). This solution is continued until the 24th hr. From the 24th hr. to the 48th hr. we administer a solution containing either 39 or 26 mEq. of sodium per L., it being obtained by mixing 300 cc. of dextrose in Ringer's lactate with 700 cc. of dextrose in water, or 200 cc. of dextrose in Ringer's lactate with 800 cc. dextrose in water. This electrolyte therapy is similar to that used in case 5. No intravenous sodium is allowed after the 48th hr. Frequent oral feedings are encouraged after 48 hr. The total colloid therapy should be roughly approximated as kg. body weight × per cent third degree or deep second degree burn × 1. Colloid therapy should be withheld in the second degree burn unless the patient develops hypotension; however, in the extensive second degree burn, the plasma loss into the wound may well require the administration of plasma.

It should be remembered that these studies are pilot studies and future work will be necessary to confirm our beliefs in the above concepts; however, the author has within the past year treated two adults with a 55 per cent total burn and a 68 per cent total burn using similar electrolyte and colloid therapy, with successful results.

CONCLUSIONS

The physiopathology of the burn has been discussed with particular emphasis on plasma loss and hypotension. Case reports of nine infant and child burns have been studied from a clinical and laboratory viewpoint with varied types of treatment and particular reference to sodium intake and excretion studies. Our beliefs regarding the the treatment of burns in infants and children have been summarized.

Acknowledgment. Grateful acknowledgements are made to Norman Jacob, M.D., in charge of the laboratory, Juanita Landez, laboratory technician, and to the residents and interns at the Robert B. Green Hospital, San Antonio, Texas.

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THE INFLUENCE OF MALPRACTICE SUITS ON SURGERY*

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The concern expressed by many individual physicians and medical society executives over the increase in malpractice suits led to a survey by the American Medical Association. While only a third of the physicians polled were of the opinion that the incidence of suits had increased, there was a clear cut expression of opinion that they had increased in California, Louisiana, New York, Rhode Island, Utah and the District of Columbia. Perhaps the large awards by the courts, ranging from $100,000 to a quarter of a million dollars have thrown the spotlight on an endemic problem.

As I have listened to discussion in the surgeons' dressing rooms and hospital corridors and at committee meetings, I have wondered if these suits have any influence on surgical practice.

To obtain this information I made an informal survey of a small but diversified group which included: 4 general surgeons, 4 neurosurgeons, 2 orthopedists, 2 chest surgeons, 2 specialists in obstetrics and gynecology, 1 plastic surgeon, 3 anesthesiologists, and 1 roentgenologist.

The problems of anesthesiology and roentgenology are so closely related to surgery that the survey would have failed to give the over-all picture if they had not been included.

A majority of the men questioned have or had teaching appointments in one of our medical schools and all but one have American Board certification.

No attempt was made to secure a true cross section but merely to learn what some of our colleagues are thinking.

I talked to each individual and asked the following questions:

"Do malpractice suits have any influence on your surgery or the manner in which you practice surgery?"

"Does the possibility of a suit prevent you from performing any type of operation or diagnostic procedure?"

"In your opinion, do malpractice claims work to the ultimate good or harm of the patient?"

One orthopedic surgeon and one chest surgeon said the idea of a suit never entered their minds. A second chest surgeon stated that he bears in mind such possibility when he has an aortogram. The roentgenologist was also of the opinion that the risk of a suit made him avoid aortograms in some cases.

Several surgeons denied that malpractice suits influenced their surgery but when asked if they had given up any operation because complications might lead to a claim against them, qualified their stand.

A general surgeon has given up the Wertheim radical hysterectomy for carcinoma because of ureteral fistula which sometimes occurs. Instead, he employs simple total hysterectomy which has fewer complications but probably not as high a rate of cure.

Two neurosurgeons no longer cut the vestibular nerve for Ménière's disease since one of them had suit filed against him in the District of Columbia for damaging the facial nerve.

A third neurosurgeon turned down a severe causalgia caused by intravenous injection in the anticubital space too close to the median nerve. The patient's personality and attitude toward the doctor who made the intravenous injection led him to believe that a suit was already in the offing. She was so uncooperative he felt that anything less than a perfect result would involve him along with the physician.

As in the causalgia case, the personality of the patient and other circumstances receive more consideration. A general surgeon was consulted by a man who had had two previous operations for inguinal hernia and alleged that he had developed testicular atrophy as a result of one of the operations. On learning that a claim had been made against the doctor who had done a previous operation, this surgeon commented on the recurrence rate of such hernias and declined to do another operation.

A man consulted a general surgeon because of severe cramp-like pain in the feet and legs. An

Presented during the Southeastern Surgical Congress March 10–13, 1958, Baltimore, Maryland.

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aortogram revealed advanced obliterative arterial disease. Sympathetic block relieved most of the patient's pain and sympathectomy was considered for palliative relief. In discussion with the family their attitude and questions led the doctor to believe that anything short of complete relief would lead to disappointment and criticism so he decided not to operate.

All three anesthesiologists attribute the decline in use of spinal anesthesia to malpractice suits. Since the doctrine of *res ipsa loquitur* has been applied to the neurologic complications, the risk is too great.

The recent scare about the dangers of radiation has led to changes in this field of therapy. The roentgenologist no longer employs x-ray therapy in benign conditions but uses it only in malignancy.

A general surgeon has given up radium therapy in all cases of uterine bleeding and substituted hysterectomy. Obstetricians and roentgenologists have reduced the use of x-ray in pelvimetry. Doctors get more x-rays of the skull after head injury, more x-rays of the ribs after chest injury, and more x-rays for sprained ankle than necessary for purely medical reasons because they are dictated by court decisions.

As we review these procedures perhaps the same thought occurs to all of us. These changes might have occurred in the natural course of events and have merely had attention focused on them by malpractice claims. While this may be true, the choice of a procedure should be based on the doctor's judgment as to what is best for the patient, not on what procedure has the least reprisal rate. To paraphrase George Bernard Shaw in his delightful comedy "The Doctor's Dilemma" should a doctor be put in a position of saying,

"Is it better for a patient with Ménière's Disease to have vomiting and dizziness or for me to risk being sued?" In choosing between inhalation, an intravenous and a spinal anesthetic, when the anesthesiologist attempts to decide which is safest and which carries the least risk, he should mean for the patient not for himself.

To the question, "In your opinion do malpractice claims work to the ultimate good or harm of patients?," a majority considered the harmful effects outweighed the good but stated that some benefit will probably be derived from seeing ourselves as others see us, and bringing to our attention errors or incidents that lead to complaints. It will turn us to the study of the malpractice situation as we study other medical problems. We will reevaluate our procedures and attempt to improve them.

The most uniform comment was that surgeons spend more time before operation explaining to a patient what they are going to do and discussing the possible complications and risks involved. They talk to the family and the patients and, in an elective operation, the patient helps make the decision.

If any complications develop after operation, they call a consultant, first, to get the benefit of his opinion and second, to share the responsibility.

The harmful effects are less tangible but it was the consensus of opinion that malpractice claims tend to retard medical progress. This is well expressed by Louis J. Regan in his book, "The Doctor and Patient and the Law."[1]

"There can be no question but what the destructively critical attitude of an ever increasing number of people, and the rising incidence of malpractice claims consequent thereon, puts a definite brake on medical progress, and seriously burdens and injures the individual physician-patient relationship."

One of my colleagues suggests that the increase in claims against physicians represents a social change which came in with the New Deal, a seeking after greater security. Another said, "When I was a boy if I fell out of my neighbor's swing or slipped on his walk, it was because I was clumsy. Now, the neighbor is liable for negligence. If a friend picked me up in his auto and we had a collision, it was merely unfortunate. Now, the driver is held liable."

During the last year I have examined many patients injured in auto accidents. All but one claimed he was run into by someone else. I spoke of this to another neurosurgeon. According to his records all of his patients were involved in accidents through no fault of their own, including two who collided with each other.

CONCLUSIONS

1. Malpractice suits do have an influence on surgery.

2. Only a limited number of procedures have been affected by the threat of suit.

3. More time is devoted to discussion with the patient of the probable risks and possible complications before operation is undertaken.

4. Additional tests, consultations, and x-rays have resulted in increased medical expense to the patient.

5. Malpractice claims have some beneficial effects on patient care in that they are leading to study and re-evaluation of medical and hospital procedures.

6. The harm resulting from malpractice claims outweighs the benefits as they tend to retard medical progress and set up a barrier to satisfactory doctor-patient relationship.

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BOOKS—AND THE EDUCATION OF A SURGEON*

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I want to thank the officers and members of The Southeastern Surgical Congress for the high honor they conferred upon me by electing me president. The work entailed in this was not difficult, so the office in reality has been one of honor and pleasure.

One of the advantages of being president is that his talk does not have to be entirely scientific. I have chosen a subject which is quite familiar to all of us—books. A doctor lives with books and uses them far more than most men throughout his life. Seldom do we stop to consider their real significance, their history, their importance. Today they surround us on every side, coming out in hundreds and thousands as new books, as new editions of old books, and as monthly journals to be bound later into volumes. This has not always been so, and as a matter of fact, books as we know them are relatively recent. Long ago, even before he was literate, man had attained civilization, in that he had significant social customs, was able to produce by cultivation, and had erected magnificent buildings. But he could not write and transmit his knowledge from one generation to another. True, there were a few scholars in Egypt who wrote on rolled papyrus, in Assyria who chiseled out the glories of conquests by their kings, with much boastfulness of their power and supremacy. But these were rarities and it was only with the advent of Greek and Roman civilization that writing became more common. Papyrus began to disappear with the advent of paper production in the Eighth and Ninth Centuries. Books were produced with greater frequency and in greater numbers; still books did not circulate as there were too few of them. In the Middle Ages they were bound and chained to shelves in the abbeys or in the palaces of the nobility and they were far too dear for the average man to see or use or understand.

In the middle of the Fifteenth Century—about 1454 or perhaps a little before that—a new process was used by a man named Gutenberg in Mainz, Germany. This was the start of printing in movable type. This process had been used by the Chinese for several centuries but it was never freely employed by them because the number and complexity of the descriptive characters in Chinese comparable to our alphabet gave printing such small advantage over the brush method of writing. When, however, printing came to the Western World the conditions were different. The alphabet had only 26 letters and type could be made and shifted with ease and efficiency justifying the conversion from the time-consuming manual method to a machine. Only 300 copies of the Gutenberg Bible were printed and of these only 45 are known to exist today. It is a peculiar thing that the Gutenberg Bible was made to look like the illuminated manuscripts of the Middle Ages. One reason, it is said, was because the printers did not want to disclose their new process. Large decoration letters were used to start chapters and the margins of the book were illuminated or illustrated beautifully, with designs in color. Many of the first productions after the invention of printing were artistic books, and they come down to us as collector's items, some of them extremely rare, now only museum pieces. Like the Gutenberg Bible, books at that time were produced in very limited numbers, and time and loss and destruction have diminished these, so that they have become very valuable and rare. William Caxton was the first printer to produce books in English. He was an Englishman who learned his trade in Holland.

Now, consider the difference between the Greeks and the Romans who left us a wonderful literary heritage and the French and the German and the English people of the same period. There were hundreds of thousands of Frenchmen and Germans in those days. They eventually overran Italy, but Julius Caesar tells of cities on the primitive sites of some of the large French cities of today where as many as 200,000 people were congregated. We have from them today, nothing but silence, simply because they did not write. They left no books. All we know about them was

* Presidential Address presented during the Baltimore Assembly of The Southeastern Surgical Congress, March 10–13, 1958, Baltimore, Maryland.

what was observed by the nations who did write at that time. Caesar realized the danger from these hordes and he was constantly battling in the summer and constantly alerted in his winter quarters to restrain them from getting organized and descending upon the Roman empire. No knowledge was transmitted by them in books. Songs and sagas and traditions eventually were recorded but of course much was lost.

Now about 100 years after Gutenberg initiated printing with movable type a very important medical book was printed. In 1543 Vesalius with the artist, Jan Stefan van Kalkar, and the printer, Oporinus of Basel, produced a beautiful volume "de Humani Corporis Fabrica." This was a great step forward in medical literature and education. When one compares this book with *Canano* and the crude inaccurate anatomies which preceded it, one is impressed, not only with this book and with its accuracy, but with *books*—how they can set down knowledge and transmit it from one generation to another to be added to, but never to be lost completely. It does something to a doctor to hold in his hands, as I have, a First Edition of the *Fabrica*. Vesalius gave these to his students and perhaps he handled this very one. Then there are the *Oeuvres* of Ambrose Paré collected and published towards the end of his life in 1575 and in which he aspired to collect the surgical knowledge of his day. When one looks into this volume in comparison to our modern surgical texts he can appreciate the tremendous advances which have been made, the ignorance with which they labored in past generations, and the accretion of knowledge from one author and generation to another down through the ages.

Among the deficiencies in present day medical education are suitable courses in medical history. Too few of the students know the great names of the past; too few of them have had a chance to see some of the early literature; to hold in their hands at least a facsimile of Harvey's *de Motu Cordis*; to see the illustrations of Galvani, stimulating muscle with electricity back in 1790; to know the now feeble effort of John Hunter on aneurysms, tying the vessel in what is known today as Hunter's canal, proximal to the popliteal artery; and even to know our modern literary greats, Sir William Osler, Harvey Cushing and others.

Naturally some men will have a greater interest in books and a greater love for them than others, and some will finally become so fascinated by them that they stimulate him to collect them. Taste influences one in book collecting. There are all sorts and varieties of book collecting. One may simply collect beautifully bound books or he may collect one author, like a surgeon I know who told me that for ten years he had the first bid on every First Edition of Goldsmith which appeared in various bookstores in London. Or one might care to collect books published by famous presses, like Aldene Press of Venice which in 1495 began to make available the printed Latin classics, or the more recent Kelmscott Press initiated by William Morris in 1891. Books from this press are beautiful and very costly. Even though modern, a *Chaucer* published by the Kelmscott Press, was recently quoted at £ 500 by an English bookstore. And almost simultaneously a New York book dealer quoted one at $1,100.00.

Other books appeal to a collector because they are the first editions or because they are illustrated by a famous artist like Cruikshank or Rowlandson or by the authors like Kate Greenaway and Thackeray. Books illustrated by Rackham and Szyk have become collector's items even though they are contemporary with our time. It is natural for one who collects books to have certain strong preferences among the authors. Not unlikely included among them will be Samuel Johnson and Boswell. One may spend pleasant times with them and with their friends, Garrick and Goldsmith and Joshua Reynolds, at the meeting of the club at the Mitre Tavern. Furthermore, admirations for old Sam may be so great that one will acquire a first edition of Boswell's *Life of Johnson* which was published in 1791. Now a book collector when he rereads this finest biography ever written will probably use a different edition, but appreciation of the greatness of this book makes him want to own the *first* edition. It is also the love of the characters, the beauty of the literature and the admiration for the excellence that makes one want 'to invest' in scarce first editions and pay bonus prices for them. But there will never be more first editions of the fine literary heritage of mankind and there will be more lovers and admirers of the great authors.

I know old Sam Johnson and I love and admire him. I have read all sorts of books about him and his friends. The recent discovery of Boswell's Journals and the fine volumes so far released are a most interesting extension of this literary period.

One who loves literature acquires many friends—Tolstoy and Dostoevski and Manzoni and Thackeray and Alfonse Daudet and Marcel Proust and Pierre Loti and Shakespeare and Shelley and the immortal Keats (who by the way like Goldsmith studied medicine) and the modern counterpart of Keats, Dylan Thomas whose scintillating brilliance finally consumed itself; and Conrad and Lafcadio Hearn and Jack London and Herman Melville who wrote *Moby Dick*, the most profound novel ever written. Then there are the Greeks and the Latins—Homer, Lucian, Epictetus, Marcus Aurelius, Julius Caesar, Virgil, Plutarch and Saint Augustine. Some of these friends speak more interestingly than others; some more beautifully.

I did not mention Dickens. Though I have his works I would not pay for one of his first editions. Why? Because I have had to force myself to read him. Yes, others like and admire Dickens. It is all a matter of taste. He is a chatterbox, a reporter. His literature is not beautiful like the language of Poe and the seascapes of Conrad. He is not profound; just superficial. On the other hand Dostoevski writes about the souls of men. Ishmael in *Moby Dick* beautifully contemplates the profundity of the meaning of life and every little thing to him illustrates the seriousness of it all. Listen to this when he is saying a harpoon line may drag a man overboard, "So the graceful repose of the line as it silently serpentines about the oarsman before being brought into actual play.... This is the thing which carries more of true terror than any other aspect of this dangerous affair.... but it is only when caught in the swift sudden turn of death that mortals realize the silent subtle, ever present perils of life. And if you be a philosopher, though seated in the whale boat, you would not at heart feel one whit more of terror, than though seated before your evening fire with a poker and not a harpoon by your side." This magnificently expressed thought was engendered by his description of the rope attached to the harpoon.

Do you know Lafcadio Hearn? He lived in New Orleans from 1877 to 1887 and he wrote most beautiful language about a variety of things. He was a friend of Doctor Matas and dedicated a novel, *Chita*, to him. It was published in 1889. In the period he lived in New Orleans he wrote many letters back to his friend, Krehbiel, in Cincinnati, and in some of them told about the brilliant young Doctor Matas. Hearn is difficult to classify but perhaps he belongs closest to the French Romanticists. Next to the extremely rare *Gombo Zêhbes*, which is a translation of proverbs from the Creole, *Some Chinese Ghosts* is perhaps the dearest collector's item. It was published in 1887. The delicacy and beauty of Hearn's literary style is seldom equalled. His best known works are the interpretation of Japanese life and culture. He spent many years in Japan. He married a Japanese, began to support her whole family and never left that country. Lafcadio Hearn died in 1904 and his friend, Matas, an equally great man in another sense, passed away recently in the fall of 1957. I love to read Lafcadio Hearn at night after a hard day of great responsibilities. Somehow I hardly believe the stories or implications I hear even today from the members of the staff of the Times-Picayune that he was dirty and most immoral. His beautiful writing and delicate thoughts make me think that whatever his weakness he was a great and a good man.

I have a first edition of an author whom, if you knew personally, you would love—Charles Lamb. *Elia*, collected in book form, first was published in 1823. I acquired this first edition in London. To be a true first edition it must have "Fleet Street" with no number in front of it on the title page. Later issues of this first edition had a street number in front of "Fleet Street." Charles Lamb was a genius. He worked many years as a clerk for the East India Company. He cared for an invalid sister. He had the gentlest, kindliest thoughts any man could ever have, and he wrote them down in beautiful and fascinating language, in such contemplations (I do not like to call them essays because to me essays connote a certain element of harshness) such as "The Praise of Chimney Sweepers" and "A Complaint of the Decay of Beggars in the Metropolis." He never married but he wrote the beautiful "Dream Children; A Reverie." His literary style is gentle and loving and it is faultless. His company pensioned him after he became a great literary figure and he wrote of his gratitude and the happiness of his last working day in "The Superannuated Man." He is an author who makes attractive and interesting even the most commonplace people for it is with people he is most interested and he discerns with warmth and love the good points of their characters.

I would like to tell you about many more of my

friends, more about how books impress and interest me but it could easily become projected and disinteresting to you. Permit me to name one more if for no other reason, because of what she says about books. Emily Dickinson belongs truly among the great. She lived in a dream world of flowers and birds, in the romance of distances, in sunshine and far away on the mysterious sea. She says of books—

> There is no frigate like a book
> To take us lands away.
> Nor any coursers like a page
> Of prancing poetry.
> This traverse may the poorest take
> Without oppress of toll.
> How frugal is the chariot
> That bears a human soul.

Books are cultural; books may be works of art and they influence the lives of surgeons far more than is generally appreciated. They can be diversions as well as sources of information. I hope you will accept this little talk as an allusion to a hobby and a tribute to one of the most helpful tools in a surgeon's career—books.

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Editorial

BURIED TALENT

The dubious distinction of becoming a senior member of the surgical fraternity has some minor compensations; they include broadening of perspectives and more courage to express unorthodox opinions. From the vantage point of nearly forty years as a surgeon and educator, I view the national scene with mixed feelings. I find much that is praiseworthy, because enormous progress has been made in both the science and the practice of surgery. On the other hand, it is equally obvious that many defects and difficult problems remain.

For one thing, I am deeply disturbed by the sight of many eager, idealistic, young men who, after a splendid year as senior resident, are thrust abruptly into a surprisingly uncharitable, selfish world of professional competition and public criticism. From a feast of responsible major surgery, wherein the resident condescendingly turns over hernias, appendices, gall bladders, and other "small stuff" to assistant residents, he is suddenly reduced to a starvation diet of surgical crumbs. From warm comradeship with members of a distinguished university hospital staff and a position of considerable importance and influence, many an ex-resident is demoted to the position of a lonely, bewildered, disillusioned young doctor, stumbling along, trying to start a practice. In order to survive, he may have to turn for a time to general practice for which he is ill prepared. Many years may pass, especially in larger cities, before he is able to develop and limit himself to the sort of practice for which he was trained.

Some young surgeons are embittered by the experience; by the observation that well-established colleagues, although worn and harried by overload, tend to hang on to every bit of practice that comes to them, including unneeded industrial and insurance contracts; by difficulties in securing suitable office space or active membership on hospital staffs; by being accorded privileges in the course of time only to be subjected to further restraints of periods of probation and supervision that take no account of documented records of prolonged surgical training in well-known surgical centers or certification by specialty boards; by observation that large amounts of operative work are being done by persons with limited training and experience.

There is something essentially wrong with that whole arrangement. It is not fair to the residents or to the men who have devoted years to their training. It is not fair to the public which should be given the benefits of the best available surgical services. It is a waste of manpower and of highly trained competence. No first-class corporation would think of going to great trouble and expense to train experts only to shelve them for several years.

The existing system has provided fertile soil from which have sprung fee-splitting, ghost surgery, and other open and hidden malodorous practices. The frustrated young surgeon, often in debt, craving some of the "good things of life" that he and his family have been denied through long years of preparation, is tempted to overcharge for his services. And when finally he does acquire a good practice of his own, he tends to perpetuate an ungenerous attitude toward oncoming young surgeons.

I do not pretend to have the correct answer to this vexatious and complex problem. A satisfactory solution may require the corporate wisdom and combined efforts of the whole professional fraternity. I shall make bold, however, to submit a few suggestions in the hope that they will stimulate more widespread and serious consideration of the matter.

1. It is high time that the problems were faced more frankly and openly by individuals and groups within the medical profession. The existence of an obvious situation that prevents maximal contribution to the public good and seeming indifference to the situation by the medical profession are the very conditions that invite the advent of socialized medicine.

2. Regulation of operating privileges by individual hospital boards and staffs is a healthy trend. In rural areas and small communities this aspect of the problem might not arise at all, but in cities where there are many physicians and a variety of trained surgeons, the staffs and governing bodies of local hospitals are the agents best

suited to adapt institutional regulations and restrictions to local needs and to available professional personnel. In this regard, hospitals must not only consider the interests of visiting staffs but also ensure that the public for which they are responsible can obtain the best available medical and surgical care.

3. An obligation to refer major surgical work to qualified surgeons should, in my opinion, be matched by a general reduction in surgical fees. By and large, scales of surgical fees are too high. If the average surgeon, including the qualified young surgeon starting practice, were able to limit himself strictly to surgical patients, referring all others to nonsurgical colleagues; if he were able to do more major operations per month than he now does, and at the same time reduce his professional fees for each operation proportionately, he would still make a good income, his relations with his nonsurgical colleagues would be happier, and he could devote himself more fully to the sort of work for which he is best suited.

4. In health and hospital insurance, an overemphasis on surgical fees needs to be corrected. It is manifestly unfair when the hospital business office, the surgeon, the anesthesiologist, and the radiologist get paid according to relatively generous schedules of fees, whereas the diagnostician, the internist, the pediatrician, and the family doctor receive comparatively meager compensation for the time, indispensable skill and devotion they contribute to the case.

5. I would be strongly opposed to any governmental or organizational attempt to regulate the numbers of surgeons or surgical specialists which may be trained. Any such policy would violate a cherished principle of free enterprise. It would be better to leave that phase of the problem to the slow but ultimately effective law of supply and demand.

6. The present policy should be continued, however, whereby the profession itself limits accreditation for training of surgeons to qualified hospitals with staffs which are willing to devote needed time and effort to the purpose.

7. A general policy should be adopted whereby the public will be enabled to discover, without difficulty or embarassment, which physicians are qualified specialists; otherwise, certification by specialty boards fails of its primary objective. A surgeon, for example, who has earned the distinction of being a diplomate of the American Board of Surgery, might be permitted and encouraged to place A.B.S. as well as M.D. after his name in the telephone book listings. So also with diplomates of other Boards. The public would soon learn that the doctor in question is thereby officially certified as being qualified in his specialty. It is a ridiculous state of affairs when a citizen, seeking good medical care, calls the County Medical Association Office only to be told that it is not permitted to give out information whether a given doctor is or is not a qualified specialist.

It is only fair to point out that the existing situation did not come about through design or the self-interest of any particular group. It just developed. It has both advantages and disadvantages. It benefits the busy, established surgeon; it is hard on the beginning surgeon; it appeals to many general practitioners because it offers them additional income from occasional surgical fees; it spawns ghost surgery and raises malpractice insurance rates. Most unfortunately, it denies to the public much expert surgical talent which has been provided largely at public expense. The medical profession would be well advised to do something about this matter voluntarily rather than wait for the issue to be forced by worsening of public relations or threat of governmental regulation.

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